Frontal Lobe Function and Performance Monitoring following Total Sleep Deprivation

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Abstract

Imaging studies have shown reduced frontal lobe resources following total sleep deprivation (TSD). The anterior cingulate cortex (ACC) in the frontal region plays a role in performance monitoring and cognitive control; both error detection and response inhibition are impaired following sleep loss. Event-related potentials (ERPs) are an electrophysiological tool used to index the brain's response to stimuli and information processing. In the Flanker task, the error-related negativity (ERN) and error positivity (Pe) ERPs are elicited after erroneous button presses. In a Go/NoGo task, NoGo-N2 and NoGo-P3 ERPs are elicited during high conflict stimulus processing. Research investigating the impact of sleep loss on ERPs during performance monitoring is equivocal, possibly due to task differences, sample size differences and varying degrees of sleep loss. Based on the effects of sleep loss on frontal function and prior research, it was expected that the sleep deprivation group would have lower accuracy, slower reaction time and impaired remediation on performance monitoring tasks, along with attenuated and delayed stimulus- and response-locked ERPs.

In the current study, 49 young adults (24 male) were screened to be healthy good sleepers and then randomly assigned to a sleep deprived (n = 24) or rested control (n = 25) group. Participants slept in the laboratory on a baseline night, followed by a second night of sleep or wake. Flanker and Go/NoGo tasks were administered in a battery at 10:30am (i.e., 27 hours awake for the sleep deprivation group) to measure performance monitoring. On the Flanker task, the sleep deprivation group was significantly slower than controls (p's <.05), but groups did not differ on accuracy. No group differences were observed in post-error slowing, but a trend was observed for less remedial accuracy in the sleep deprived group compared to controls (p = .09), suggesting impairment in the ability

to take remedial action following TSD. Delayed P300s were observed in the sleep deprived group on congruent and incongruent Flanker trials combined ($p = .001$). On the Go/NoGo task, the hit rate (i.e., Go accuracy) was significantly lower in the sleep deprived group compared to controls ($p < .001$), but no differences were found on false alarm rates (i.e., NoGo Accuracy). For the sleep deprived group, the Go-P3 was significantly smaller ($p = .045$) and there was a trend for a smaller NoGo-N2 compared to controls ($p = .08$). The ERN amplitude was reduced in the TSD group compared to controls in both the Flanker and Go/NoGo tasks. Error rate was significantly correlated with the amplitude of response-locked ERNs in control ($r = -.55$, $p = .005$) and sleep deprived groups ($r = -.46$, $p = .021$); error rate was also correlated with Pe amplitude in controls ($r = .46$, $p = .022$) and a trend was found in the sleep deprived participants ($r = .39$, $p = .052$). An exploratory analysis showed significantly larger Pe mean amplitudes ($p = .025$) in the sleep deprived group compared to controls for participants who made more than 40+ errors on the Flanker task.

Altered stimulus processing as indexed by delayed P3 latency during the Flanker task and smaller amplitude Go-P3s during the Go/NoGo task indicate impairment in stimulus evaluation and / or context updating during frontal lobe tasks. ERN and NoGo-N2 reductions in the sleep deprived group confirm impairments in the monitoring system. These data add to a body of evidence showing that the frontal brain region is particularly vulnerable to sleep loss. Understanding the neural basis of these deficits in performance monitoring abilities is particularly important for our increasingly sleep deprived society and for safety and productivity in situations like driving and sustained operations.

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Frontal Lobe Function and Performance Monitoring following Total Sleep Deprivation

The effects of sleep deprivation on human performance were first reported in the late 1800's by Patrick and Gilbert who studied three men in their mid-twenties for 90 hours of continuous wakefulness (1896). Patrick and Gilbert observed that the three men often encountered uncontrollable lapses in attention that sometimes resulted in micro-sleeps with immediate rapid eye movement (REM) sleep onset. They also reported significant fluctuations in daytime performance on tasks that measured reaction time, memory and time discrimination. All participants had intense recovery sleep characterized by what the authors described as deeper, longer sleep. More than a hundred years after that seminal study, research has shown that sleep loss reliably leads to deficits in reaction time, attention, memory and mood states (for reviews, see Hart, Buchsbaum, Wade, Hamer & Kwentus, 1987; Durmer and Dinges, 2005). Despite a vast literature describing the effects of sleep loss on human performance, the underpinnings of the neural circuitry driving performance instability are still not well understood (Dinges & Kribbs, 1991). Given that sleepiness is a major public health concern with increased incidence of shift work, sleep disorders (Committee on Sleep Medicine and Research, 2006), and the aging demographic, it is imperative that brain mechanics and individual differences driving performance while sleepy be better understood. This thesis reports data on the behavioural and neurophysiological effects of total sleep deprivation (TSD) on performance monitoring using error monitoring and response inhibition tasks from a larger laboratory study of 34 hours of TSD designed to investigate frontal lobe function and emotion regulation.

Brain Imaging studies of TSD

Brain imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) are useful in indentifying key areas of the brain that are vulnerable to sleep deprivation. The earliest PET studies on sleep deprivation showed that a reorganization of cerebral metabolic activity occurred after TSD. Wu et al. (1999) observed decreases in metabolic activity of temporal lobes and increases in the visual cortex, as well as decreases in localized areas of the thalamus, basal ganglia and cerebellum after 32 hours of TSD. A year later, Thomas et al. (2000) showed significant decreases in global metabolic glucose rates and localized changes in cortical and subcortical structures such as prefrontal and posterior parietal sites, as well as the thalamus, that progressed over 85 hours of sleep deprivation.

An fMRI study conducted by Portas, Rees, Howseman, Josephs, Turner and Frith (1998) showed increased activation in the ventrolateral thalamus in participants with the lowest levels of arousal on an attention task; cerebral activation changed as a function of arousal. Other early MRI studies using arithmetic working memory (Drummond et al., 1999), verbal learning memorization (Drummond et al., 2000) and divided attention tasks (Drummond et al. 2001), showed that brain regions typically associated with cerebral activation (i.e., prefrontal cortex) were also activated in the sleep deprivation condition in addition to more widespread prefrontal areas as well as bilateral inferior and superior parietal cortices. A recruitment discrepancy was observed between the verbal learning and arithmetic working memory tasks which the authors concluded as differences in task demands and may be a compensation or recruitment strategy to help deal with subjective sleepiness or performance impairments (Drummond et al., 2001).

Lapses in attention are one of the greatest hallmarks of sleep loss. Drummond, Bischoff-Grethe, Dinges, Ayalon, Mednick and Meloy (2005) have shown that differential brain regions activate and inactivate depending on the RTs to specific stimuli in a psychomotor vigilance task. Under fast RT conditions, areas such as the putamen, inferior parietal lobes, DLPFC and several medial or bilateral motor regions showed an increase in activation; these areas are thought to represent sustained attention and motor output. Under slow RT conditions, frontal and medial cingulate gyri regions became activated; these are thought to represent the so-called 'default mode network' (Raichle, MacLeod, Snyder, Powers, Gusnard, and Shulman, 2001). This area of the brain is activated when individuals are not cognitively engaged; however, when situations arise that require problem solving, resources may be reallocated away from the default network to the specific brain regions that are responsible for proper cognitive functioning. Drummond et al. (2005) showed that under periods of prolonged wakefulness individuals may be able to perform equally well as their control counterparts; however, lapses in attention will occur and differential brain regions turn on and off depending on the attentive state.

Later, using fMRI, Yoo, Gujar, Hu, Jolesz and Walker (2007) showed that those in a sleep deprivation condition expressed a greater than 60% increase in magnitude of amygdala activation in comparison to the control condition to negative pictures from the International Affective Picture Set (IAPS). They also showed regions of amygdala co-activation and found that there were significantly weaker connections between the medial prefrontal cortex (MPFC) and the amygdala in the sleep deprived compared to the rested

controls. Yoo et al. (2007) suggested that a failure in top-down prefrontal control may result in inappropriate emotional responses due to the MPFC amygdala disconnect.

Chuah, Dolcos, Chen, Zheng, Parimal and Chee (2010) have shown increased disruptions in working memory when participants were introduced to negative valance distracters. Like Yoo et al. (2007), Chuah et al. showed increased amygdala and ventrolateral prefrontal cortex activation following sleep deprivation. They also showed the disconnect between the amygdala and the prefrontal regions in the sleep deprived condition. The cognitive-affective link in the PFC appears compromised following sleep loss; this could explain the reduced efficiency in rapid top-down judgements involving emotional information (Yoo et al., 2007; Killgore et al., 2007; Chuah et al., 2010).

Taken together, these imaging studies show that the sleep deprived brain shows differential patterns of activation and deactivation in attention networks, PFC and limbic disconnections, and periodic switching between attentive and inattentive states.

Electrophysiological studies of sleep deprivation

Compared to brain imaging techniques, electroencephalography (EEG) and event-related potentials (ERPs) have poorer spatial resolution but superior temporal resolution. Spatially, hemodynamic measures can be precise to a millimetre, while electromagnetic measures depend on the number and placement of electrodes. Temporally, the hemodynamic response is limited to seconds whereas ERPs are accurate up to one millisecond (Luck, 2005). These differences allow EEG and ERP techniques access to some research questions that brain imaging techniques simply cannot answer.

Research on the effects of sleep deprivation on arousal using EEG are documented back to 1937 when Blake and Gerard investigated EEG in three participants

following 60 hours of wakefulness. They showed that individuals deprived of sleep expressed regular wake potentials with alpha EEG prior to sleep onset. However, if the participant was left to relax, immediate changes were observed such that very large slow delta waves were generated with an immediate rebound into slow wave sleep.

The alpha EEG 8-12Hz oscillation is typically used as an index for relaxed wakefulness or cognitive disengagement (Andreassi, 2007). A study by Cajochen, Brunner, Krauchi, Graw and Wirz-Justice (1995) reported that subjective fatigue correlated significantly with increase EEG power in the theta/alpha range. EEG was recorded for four minutes at 3, 10, 27 and 34 hours of extended wakefulness. They concluded that there may be a homeostatic drive to regulate theta/alpha EEG when wakefulness is prolonged. Using multiple channel EEG recordings, Lorenzo, Ramos, Arce, Guevara and Corsi-Cabrera (1995) showed greater alpha and theta EEG power during performance errors and slowed RTs on a vigilance task. Lorenzo et al. (1995) also observed an increase in beta activity which they suggested represents an increase in effort to remain awake rather than as an index for increased arousal. This increase in beta activity may be short lived as the effects of total sleep deprivation typically produce lapses in attention; increased effort and motivation may compensate behaviourally for only short periods of time (Corsi-Cabrera, Arce, Ramos, Lorenzo, & Guevara, 1996).

Various stages of information processing can be captured using ERPs. This technique makes ERPs ideal for measuring the brain basis of cognitive dysfunction following sleep deprivation. ERPs allow researchers to pinpoint the alterations in underlying brain function (i.e., whether it is early sensory encoding or late cognitive processing) when experimental manipulation is introduced (Luck, 2005). The ERPs

investigated in this thesis include stimulus-locked potentials (N1, P2, N2, P300) and response locked ERPs to error monitoring and response inhibition tasks (ERN and Pe).

Stimulus-locked attention ERPs

Impairments in long latency ERPs including the N1, P2, N2 and P300 have been shown following sleep deprivation. Deficits in auditory ERPs due to sleepiness have come from studying narcoleptic patients (Broughton, Low, Valley, Da Costa, & Liddiard, 1982), as well as healthy sleepy individuals (Pressman, Spielman, Pollak, Weitzman, 1982). Narcoleptics and healthy sleepy individuals show very similar deficits in the latency and amplitude of N1-P2 and P2-N2 complexes (Pressman et al., 1982). The deficits observed in these long latency ERPs in a sleepy state, suggest an impairment in attentional networks, as many of these ERPs also change at sleep onset (Cote, Lugt, & Campbell, 2002; Colrain & Campbell, 2007), or following experimental sleep fragmentation (Cote, Milner, Osip, Ray, Baxter, 2003). These changes in early-attention ERPs may be driven by change in what Näätänen (1982) has termed the "Processing Negativity" (PN); an attention-related component that has an influence on both the N1 and P2 ERP components. Inattentiveness results in a smaller PN; less negativity will yield a smaller N1 and a larger P2. The N2 and P300 will be discussed in later sections pertaining to TSD and performance monitoring.

Response-Locked ERPs during Error Monitoring Tasks

Error-related Negativity. Researchers have investigated the brain's response to errors on tasks by measuring response-locked ERPs including a frontal negative deflection and a later parietal positive deflection. The neural mechanism for human error monitoring was first reported independently by two groups of researchers (Falkenstein, Hohnsbein,

Hoormann, Blanke, 1991; Gehring et al., 1993). The two groups used different names to describe the underlying neural correlates of human performance monitoring (Error-related Negativity (ERN) in the case of Gehring et al. (1993) and Error-negativity (Ne) in the case of Falkenstein et al. (1991)). Although the groups of researchers have slightly different interpretations of the ERP, they have generally concluded that the ERP is associated with the performance monitoring system.

Falkenstein et al. (1991) and Gehring et al. (1993) have described the appearance of the ERN during erroneous responses typically to speeded choice reaction time tasks. The ERN is typically largest over frontal and central midline electrodes and is represented by a sharp negative deflection in the EEG with a magnitude of approximately 10 μV usually peaking 100 ms after erroneous responses (Falkenstein et al.1991; Gehring et al. 1993). Falkenstein et al. (1991) has hypothesized that the ERN is elicited during an automatic mismatch between the overt response and the intended response. Another widely accepted view for the function of the ERN is its role in conflict detection rather than error detection per se (Botvinick et al. 1999). van Veen and Carter (2002a) have suggested that speeded reaction time tasks produce two streams of information processing at the exact same time, the correct response and the incorrect response. The moment of greatest conflict is the moment both streams are equally activated. Only when the "mismatch" occurs does the peak of the ERN follow. van Veen and Carter (2002a) also state that incorrect responses are partially contributed by partial stimulus analysis. They suggest areas of the brain responsible for generating the ERN are also active during times of high stimulus conflict; thus ERNs could potentially be generated, in part, to correctly identified trials. The conflict detection hypothesis has very little credit as more

recent research (Masaki & Segalowitz, 2004; Burle, Roger, Allain, Vidal, & Hasbroucq, 2008) has shown that the ERN represents a graded error-detection process. Masaki and Segalowitz (2004) showed using arrow, arrow-Simon and arrow-orientation tasks that manipulating stimulus conflict resulted in graded ERN responses (verified by recording EMG from both the correct and incorrect hand) from partial errors. They went on to show that the ERN to partial errors was smaller (probably because it is not a true error) and peaked faster than overt errors. The earlier and smaller ERN to partial errors allows for response correction before the overt error response is executed.

Since the magnitude of the ERN is typically observed fronto-centrally, researchers have hypothesized that this ERPs generation might arise from a specific area of the frontal lobe. A brain area known as the anterior cingulate cortex (ACC) is located on the medial surface of the frontal lobe (van Veen & Carter, 2002a) and was first hypothesized to represent attentional recruitment. The ACC has been shown to increase in activation after erroneous responses (Carter, Braver, Barch, Botvinick, Noll & Cohen, 1998). Interestingly, the ACC also increases in activation to correct trials with high levels of conflict. Carter et al. (1998) suggested that the ACC comes online when errors are more likely to occur. This activation in the ACC in the absence of an overt error may suggest the ACC represents global performance monitoring including both error and conflict detection. The ACC is divided into two subdivisions: the dorsal cognitive and ventral affective subdivision. A recent study by Kanske and Kotz (2011) found that the ACC operated differentially to conflict when it received affective information and suggests that emotion input can enhance the speed of conflict processing. They suggest

the dACC processes conflict regardless of salience, whereas the vACC only comes online when emotional information is present.

The task typically used to generate errors and high levels of conflict is a task designed by Erikson and Erikson (1974) known as the Flanker task. During the Flanker task, participants are told to respond to a centrally presented target while ignoring a string of flanking stimuli. Participants are instructed to respond with left hand or right hand (usually index finger) button presses. The RTs and error rates to trials considered congruent (i.e., identical target and flankers e.g., HHHHH) are typically quite low, whereas incongruent trials (i.e., different flanker and target e.g., HHSHH) result in the highest error rate and slowest RTs because the incongruent flankers compete with the central intended target. These incongruent trials produce response conflict and according to van Veen and Carter (2002a), the need for control during these trials is high. Although the Flanker is the most commonly used task to assess error monitoring, theoretically it is possible to elicit the ERN using any task for which there are errors and there is an awareness of those errors.

Botvinick et al. (1999) showed that trials with the greatest response conflict induced the greatest dorsal ACC activity and Carter et al. (1998) showed increased ACC activity to error detection. Both high levels of conflict and error detection induce ERNs and therefore have been hypothesized to be generated in the vicinity of the ACC. van Veen and Carter (2002b) have shown using source localization that two ERPs (the N2, which is evident in stimulus-locked trials with high degree of conflict, and the ERN, which is generated after error responses), were modeled as having a shared generator in the caudal ACC.

In addition to the ACC, some researchers have found evidence for the ERN generating from the supplementary motor area (Herrmann, Römmler, Ehlis, Heidrich & Fallgatter, 2004; Dehaene, Posner, & Tucker, 1994). Both groups used source localization techniques and were able to pinpoint a generator in the SMA. Some researchers argue against the SMA as a generator of the ERN based on the underlying neuronal orientation. Only within the cingulate sulcus do the pyramidal cells orient themselves in a way that the ERNs negativity can be largest (Holyroyd & Coles, 2002).

A neuronal model has been proposed by Holroyd and Coles (2002) that involves the dopaminergic mesencephalic system. The basal ganglia and a specific portion of frontal cortex receive neural projections from areas including the substantia nigra, ventral tegmental area and pars compacta. Additional projections from limbic structures like the amygdala have also been shown to project to the ACC. Limbic projections have a variety of influences on motor activity that are largely driven by motivational control. This model has helped shape Holroyd and Coles' (2002) reinforcement learning hypothesis. They suggest that whenever an ERN is generated, it is a result of a negative reinforcement signal sent to the rostral cingulate zone via their proposed mesencephalic dopamine system. This dopamine signal is then able to modify and shape future performance based on endogenous feedback.

Error Positivity. Over the last two decades nearly all the attention in the performance monitoring field has been paid to the ERN. A small amount of literature has presented information on the ERP following the ERN known as the error positivity (Pe). The error positivity is a positive deflection that peaks following the ERN and usually is captured within the 200 to 400 millisecond interval range (Overbeek, Nieuwenhuis &

Ridderinkhof, 2005). Pharmacological treatments that target the dopamine system have shown to influence the ERN component but not the Pe component; this suggests that each component works independently and only the ERN is susceptible to alterations in the dopaminergic mesencephalic performance system (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; Ridderinkhof, et al., 2002).

Like the ERN, the Pe appears vulnerable to specific experimental manipulations. A study by Nieuwenhuis, Ridderinkhof, Blom, Band and Kok (2001) found in an antisaccade task that Pe amplitude was conditional upon whether the participant perceived and was aware of errors. The Pe was evident only when the participant was certain they responded incorrectly. They also went on to show that the Pe amplitude was largest when the trial following the error was slowed remedially. The amplitude of the Pe may also vary with error rate. Some researchers have reported a decrease in Pe amplitude with an increase in error rate (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000), whereas others have not found support for this relationship (Herrmann et al., 2004; Hajcak, McDonald, & Simons, 2003).

In a review chapter, Falkenstein (2004) summarized advancements to date and put forth theories on the function of the Pe. Falkenstein's conscious error recognition hypothesis states that the Pe is generated when a participant is aware of a mistake. The hypothesis is supported by Nieuwenhuis et al. (2001) with the antisaccade task and evidence of post-error slowing. It has also been shown that the Pe is smaller on more difficult tasks which may suggest participants make more errors and are unaware of them due to the increased difficulty (Falkenstein, 2004). Alternatively, Falkenstein's adaptation of a response strategy hypothesis is based upon remedial actions, like post error slowing,

which improves the probability of an accurate response on the next trial. Again, Nieuwenhuis et al. (2001) supports this hypothesis with data showing the association between Pe amplitude and post error slowing. Falkenstein's emotional error processing hypothesis is supported by associations between error rate and amplitude. Another piece of evidence is provided by van Veen and Carter (2002a) as they showed that some subcomponents of the Pe are generated in the rostral section of the ACC; an area that has previously been shown to have strong connections with the limbic system (Devinsky et al., 1995). Bush et al. (2000) has also labelled this area of the ACC as the 'affective subdivision'. The Pe currently does not have a definitive role due to its ambiguous nature but does seem to play a role in error processing and remedial strategy.

Sleep deprivation and error monitoring

The first study to look at the effect of sleep deprivation on performance monitoring was done in 1999 by Scheffers, Humphrey, Stanny, Kramer and Coles. In a very small sample of all male participants (n = 8), visual and memory search tasks were administered over five training days with nine overnight sessions on the final day. They reported that ERN amplitude was reduced as a function of time on task as a result of one night of TSD. The Pe was not reported; however, Scheffers et al. (1999) reported impaired remedial actions in RT in the sleep deprived condition. The tasks used in this initial study were non traditional error monitoring tasks, but nonetheless yielded an error rate of 14% which elicited ERN waveforms.

A subsequent study by Tsai, Young, Hsieh and Lee (2005) investigated a modified arrow Flanker task in 16 mixed-gender participants in a repeated measures counterbalanced design, following both normal sleep and 24 hours sleep deprivation

conditions. P300 amplitude was reduced in both congruent and incongruent stimuli in the sleep deprived condition compared to the control condition; P300 latency was stable. As well, sleep deprivation produced a larger but delayed N2 in the sleep deprivation condition. Although the response-locked ERP components did not differ in latency, ERN and Pe amplitude were both reduced in the sleep deprivation condition. Remediation to errors was corrected for as indexed in the post-error slowing and accuracy for the normal sleep condition only. The authors concluded that error detection, as well as remedial systems, are compromised following 24 hours of TSD.

Murphy, Richard, Masaki and Segalowitz (2006) used a letter Flanker task in an all female sample (n = 17) counterbalanced across a well rested state and a sleepy state (20 hours of wakefulness). They found decreased Pe and remedial behaviour in the sleep deprived group, but no change in ERN. Murphy et al. interpreted these findings as evidence that sleep deprived participants are aware of their errors, but may not 'care' about their erroneous performance. The ERN may not have been affected in the Murphy et al. study as reported previously because of the relatively subtle degree of sleepiness (i.e., prolonging wakefulness in a sample of young participants that normally retire around 12-1 am).

Immediate error correction behaviour (i.e., when an error is committed, the participants correct themselves by selecting the intended response) has also been tested following sleep deprivation (Hsieh, Cheng, & Tsai, 2007). A repeated measures design was used to test participants in a counterbalanced manner across a sleep deprivation (24 hours) and regular sleep condition. The study consisted of 16 normal healthy good sleepers with an equal number of males and females. Reaction time, accuracy rate and

correction rate were not significantly different across conditions. Overall performance and remedial behaviour did not differ between the sleep deprived and regular sleep conditions except in certain incongruent trial types; sleep deprivation produced a decrease in correction rate for stimulus incongruent trials only. Hsieh et al. (2007) reported a significantly smaller ERN amplitude in sleep deprived condition compared to the well rested condition. The Pe appeared smaller after sleep deprivation, but was not statistically significant.

Next, Hsieh, Tsai and Tsai (2009) investigated EEG correlates to post-error adjustments following TSD. In previous work (Hsieh et al., 2007), the authors had shown that explicit instruction to correct erroneous behaviour could mask TSD effects. They showed that if an error was corrected, both accuracy and RT increased on the following stimulus. This effect was not found following errors that were not corrected. They also showed that sleep deprivation induced more delta and theta activity generally, with varying degrees of beta activation depending on trial behaviour. When sleep deprived, participants typically generated very little beta at the beginning of the trial; it was largely dominated by delta activity. However, on error trials, beta increased significantly after corrected behaviour which in turn increased the accuracy and RT on the trials following the correction. Typically, increased beta and blocked alpha and theta is thought to represent task engagement (Prinzel, Freeman, Scerbo, Mikulka & Pope, 2000). Other literature has reported increased beta activity after TSD (Cabrera et al. 1996; Lorenzo et al. 1995), which has been interpreted as effort. Hsieh et al. (2009) concluded that when a trial was corrected, sleep deprived individuals generated beta to boost performance. Thus,

intentionally correcting errors may serve as a compensation mechanism to maintain performance during sleepiness.

Asaoka, Masaki, Ogawa, Murphy, Fukuda and Yamazaki, (2010) tested performance monitoring after a one hour nap to examine the impact of sleep inertia. The impact of sleep inertia on performance is similar to effects of sleep deprivation. Nine participants (7 male) underwent a counterbalanced rest versus nap condition. Participants napped from 14:00 until 15:00 and performed an arrow-orientation Flanker task for 25 minutes immediately upon awakening. No significant differences were reported behaviourally for RT and accuracy despite the nap condition participants reporting higher on subjective performance differences. There was no difference in ERN amplitude but the Pe was reduced in the nap condition due to the effect of sleep inertia. The authors stated that the low motivational significance to a mistake (a hypothesis of Pe index) may explain the over confident subjective performance on the task. Similar to Murphy et al. (2006), more extreme levels of sleepiness may be necessary to decrease awareness of errors as indexed by the ERN. The Pe may be modulated by less extreme levels of sleepiness (possibly due to changes in mood/affect as opposed to attention).

Sleep deprivation has been shown to reduce participants standard of performance (probably through effort and motivation; Kjellberg, 1975). Since motivation plays a large role in human performance, Hsieh, Li, and Tsai (2010) investigated the impact of monetary incentives on performance monitoring in sleep deprived individuals. Twenty-four mixed-gender young adult participants performed a letter Flanker in a counterbalanced repeated measures design in a rested and TSD (24 hours) state. Half of the participants received incentive for behavioural accuracy and half did not. Results

showed that monetary incentives led to a greater confidence in task performance in both rested and TSD conditions. In the TSD condition, ERN amplitude, P300 latency, and effort was impaired; these effects were masked in the incentive condition. Monetary incentives did not alter behavioural performance in accuracy and RT variability. The Pe amplitude and remedial behaviours were also resilient to monetary incentives when sleep deprived. Hsieh et al. (2010) concluded that incentives boost confidence and effort when sleep deprived, which may override some of the effects of TSD on performance.

Response Inhibition

Harrison and Horne (2000) hypothesized that the increase in social impulsivity and behavioural outbursts following sleep loss probably stem from the compromised prefrontal top down control over the normally inhibited behaviours. Response inhibition is usually tested using a Go/NoGo paradigm. In a typical task, the participant is instructed to make a motor execution to a target stimulus (Go) and to inhibit motor responses to an equiprobable or rarely occurring stimuli (NoGo; Simmonds, Pekar, & Mostofsky, 2008). Electrophysiologists can capture stimulus-locked ERPs known as the Go-P3, NoGo-P3 and NoGo-N2, which all index specific and independent cognitive processes. The ERN and Pe response-locked ERP components may also be measured, as the Go/NoGo paradigm usually induces high levels of cognitive control and therefore high error rates.

Go-P3 ERPs

The Go-P3, also known as a P3b or the classic P300 is typically largest over the central parietal electrode site, Pz (Picton, 1992). The most common view of its functional significance is Donchin and Coles' (1988) context updating hypothesis. They postulate that the P3b indexes the continual sampling of one's environment and peaks only when

the template model needs to be revised or updated; latency represents time needed to evaluate incoming information.

NoGo-P3 ERPs

Researchers have also observed a large positive wave that is generated more anteriorly following NoGo stimuli (Fallgatter, Bartsch & Herrmann, 2002). Fallgatter et al. termed this phenomenon the NoGo anteriorisation or NGA. Using Low Resolution Electromagnetic Tomography (LORETA) modelling techniques, they showed that NoGo-P3 was probably generated in the vicinity of the ACC, suggesting that in addition to the ACC contributing to conflict monitoring as reflected by N2 ERP, it also plays a role in specific inhibitory processes. These inhibitory processes are likely generated in the ventrolateral prefrontal cortex. Other evidence authors have put forth for the NoGo-P3 playing a role in inhibition was the delayed latency of the NoGo-P3 in comparison to the Go-P3. Fallgatter et al. suggested the execution of a Go response requires significantly less attention than does an inhibited response; this increase in attention corresponds to delayed processing as more resources are needed to correctly inhibit an undesired outcome. Liotti, Pliszka, Perez, Kothmann and Woldorff (2005) found similar electrophysiological results regarding children with ADHD and their performance on a cognitive control task. Children with ADHD typically have difficulty inhibiting many behaviours. Liotti et al. showed an anterior NoGo-P3 to correctly inhibited trials in controls that was reduced in children with ADHD. They termed the P3 a P3a because it was generated at frontal sites. The P3a is typically observed to rare novel events and is thought to arise from areas in the orbitofrontal cortex or the ACC (Brázdil, Rektor, Dufek, Daniel, Jurák, & Kuba, 1999). Collectively, the localization of the NoGo-P3 to

the ACC, the delayed processing to NoGo stimuli (as indexed by delayed P3 latencies) and the reductions in NoGo-P3 amplitude in ADHD children suggest strong evidence for the NoGo-P3 as an index for inhibition.

NoGo-N2 ERPs

Although there is overall agreement for what the NoGo-P3 represents, the functional significance of the N2 remains debated by researchers. A handful of researchers argue that the N2 represents an inhibition process before motor execution (Jodo & Kayama 1992; Eimer 1993; Kopp, Mattler, Goertz & Rist, 1996; Falkenstein, Hoormann, & Hohnsbein, 1999), whereas others propose it represents a specific conflict between motor execution and inhibition (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Donkers and van Boxtel 2004).

A study by Jodo and Kayama (1992) assigned participants to one of two groups, a high response inhibition (HI) group or a low response inhibition (LI) group. Those who were assigned to the HI group were instructed to respond as fast as possible and only trials under 300 milliseconds would be considered for analysis. The LI group was told to respond to each trial only after 500 milliseconds had passed. The authors surmised that when the RT limit was set to a lower value (i.e., 300 ms), participants would be rushed to make Go responses. This RT limit would make it more difficult for a participant to withhold a response to a NoGo stimulus and would presumably require increased effort and attention to respond correctly to NoGo stimuli. Results indicated that N2 amplitudes were larger to NoGo stimuli than Go stimuli; however, the N2 to NoGo stimuli was significantly larger in the HI group compared to the LI group. Jodo and Kayama (1992)

concluded that larger N2 amplitudes in the HI group were the result of increased effort and attention to inhibit NoGo responses.

Another study by Eimer (1993) investigated ERPs to a Go/NoGo task that manipulated stimulus probability and fixation precues. The precue directed participants' attention to the likely position of the following Go or NoGo stimulus. In the first experiment, participants were instructed to respond to Go stimuli only and were directed by a precue to focus in either the left or right visual field. Go trials appeared in the precue visual field 75% of the time in the first experiment and 50% of the time in the second experiment. Eimer reported that N2 amplitude was largest when Go and NoGo stimuli were equivocal and was largely influenced by precue attention. Thus, the underlying process generating the N2 was deemed to be attention driven. Therefore, according to Eimer, modulations in N2 amplitude to Go and NoGo stimuli must be considered separately in information processing; one is responsible for response activation (Go-N2), whereas the other is responsible for response inhibition (NoGo-N2).

Kopp et al. (1996) developed a strategy to study response inhibition by creating a Go/NoGo that focused on selective response priming. They manipulated an arrow Flanker task to contain congruent, neutral and incongruent Go stimuli. NoGo stimuli had a central octagon rather than an arrow that was flanked by arrows that were termed specific right or specific left trials (depending on the direction of the flanking arrows). NoGo trials also had a trial flanked by squares, which the authors termed non-specific trials. Kopp et al. found that N2 amplitude was largest for the incongruent Go condition as well as the specific right and left NoGo trials. The authors interpreted this result as

evidence for the inhibition process because trials priming incorrect responses or trials with tendencies for an incorrect response, resulted in the largest N2 amplitudes.

Additional evidence for the inhibition hypothesis comes from Falkenstein et al. (1999). They developed a study that incorporated Jodo and Kayama's (1992) forced RT hypothesis and Eimer's (1993) attention hypotheses into a cross modal (visual/auditory) divided attention Go/NoGo task with participants divided into "Good" and "Poor" inhibition performance groups. They hypothesized that if the N2 indexes inhibition, then participants with a high false alarm rates (i.e., poor performers) would have reduced N2 amplitudes in comparison to the participants with a low false alarm rate (i.e., good performers). Results confirmed the hypothesis in that those with high false alarms showed the smallest N2 amplitudes; the authors interpreted this as poor response inhibition.

Collectively, research by Jodo & Kayama (1992), Eimer (1993), Kopp et al. (1996) and Falkenstein et al. (1999) shows strong support for the N2 representing a response inhibition function. However, Nieuwenhuis et al. (2003) and Donkers and van Boxtel (2004) suggest otherwise. One of the strongest arguments in opposition of the inhibition hypothesis comes from Nieuwenhuis et al. (2003). They showed that relative frequency manipulations in Go and NoGo trials had a large impact on N2 amplitude. They had participants perform a Go/NoGo task altering the frequency of NoGo trials from 20% to 50% to 80%. They found that the amplitude of the N2 decreased as a function of NoGo stimulus frequency. When NoGo frequency was increased to 80%, Go stimuli actually generated larger N2 amplitudes than the frequent NoGo stimuli. They concluded that N2 amplitude was enhanced for low-frequency stimuli, regardless of

intended generation (Go stimuli) or suppressed motor responses (NoGo stimuli). The N2 was localized to the ACC, the area of the brain shown to be active during high levels of conflict (Botvinick, 1999). Nieuwenhuis et al. (2003) argued that the inhibition hypothesis cannot account for the increased N2 to infrequent Go stimuli. Therefore, the N2 may be interpreted in two different ways. First, it may represent a top-down inhibition role, alternatively, it may represents a mismatch mechanism where frequency of stimuli play a large role in ERP generation (Nieuwenhuis et al., 2003).

To follow up the large N2 amplitude to infrequent Go stimuli (Nieuwenhuis et al., 2003), Donkers and van Boxtel (2004) conducted a study that again manipulated stimulus frequency but also included an additional go/GO task. In the go/Go task, like the traditional Go/NoGo task, the infrequent NoGo was replaced with a coloured GO stimulus. Participants were asked to respond with a 'maximal voluntary force' to the GO stimuli. An accurate go responses was when participants applied a minimal force (25% to 50% of their maximum voluntary force). Donkers and van Boxtel (2004) replicated the findings by Nieuwenhuis et al.(2003) of stimulus frequency and N2 physiology. Further, they showed that NoGo and GO trials led to nearly identical patterns of N2 elicitation; unlike the NoGo stimuli, the GO stimuli did not require inhibition yet still led to large N2 ERPs. Taken together these results suggest the act of motor inhibition is not necessary to elicit a N2 ERP; rather, conflict detection is a more reasonable explanation for its functional significance.

Sleep Deprivation and Response Inhibition

It has been well established that sleep deprivation and sleep restriction have vast effects on frontal lobe functioning (for review, see Jones & Harrison, 2001). Only

recently has it been shown to increase impulsive behaviors, which may be elevated in men more than women (Acheson, Richards & de Wit, 2007). Studying the effects of sleep deprivation on frontal lobe functioning, and response inhibition in particular, is necessary because the behavioural literature remains equivocal and the neural correlates are unknown.

The earliest studies involving sleep deprivation and response inhibition helped illustrate the effects of sleep deprivation on response behaviour, yet had many limitations and short comings (Harrison & Horne, 1998; Fallone, Acebo, Arnedt, Seifer & Carskadon, 2001; Jennings, Monk & van der Molen, 2003). Some early studies showed that sleep deprivation led to difficulties in inhibiting congruent word endings when asked to give incongruent meaningless sentence endings (Harrison & Horne, 1998). Other studies that restricted children and adolescence of sleep showed no association between decreased sleep and response inhibition (Fallone et al., 2001). In a larger study that looked at processes of supervisory attention in general, no evidence was found associating sleep deprivation with a deficit in response inhibition or task shifting (Jennings, Monk, & van der Molen, 2003). These early behavioural studies show unexpected null results, which may be due to sampling demographics, types of tasks employed, and levels of sleep loss.

More recent sleep deprivation studies interested primarily in response inhibition, provide clearer rationales and conclusions. Drummond, Paulus and Tapert (2006) showed using a Go/NoGo task that after 23 hours of TSD, participants had an increase in false alarm rate and increased RTs to Go trials that increased until 55 hours TSD (end of TSD period). At 55 hours, TSD participants also showed a decrease in hit rate. Both

responding to the Go stimuli and the inhibition to NoGo stimuli returned to baseline following a night of recovery sleep. Cain, Silva, Chang, Ronda and Duffy (2011) found similar results with a Stroop interference task. Participants performance on accuracy and RT worsened as a function of time spent awake over 40 hours. Although these studies involve behavioural data only, it provides insight for ERP and fMRI researchers interested in the underlying neurophysiology associated with the behavioural changes caused by sleep deprivation on response inhibition.

Schapkin, Falkenstein, Marks and Griefahn (2006a) were the first group of researchers to look at electrophysiology to a Go/NoGo task during a period of noise induced sleep disturbances. Schapkin et al. (2006a) presented railway noise to 24 young participants at varying decibel levels over a period of three nights. One night classified as a 'quiet' night served as a control. Participants then performed a Go/NoGo task approximately 20 minutes after awakening; Compatible and incompatible stimuli were divided into 50% Go and 50% NoGo trials. Uppercase and lowercase words were used in this task; Incompatible stimuli were the German words ''STOPP'' as Go responses and ''DRUCK'' as NoGo responses which translate to stop and press respectively. The incompatible trials induce interference as the uppercase word STOPP implies withholding a response, and DRUCK implies responding. Participants reported an overall decrease in subjective sleep quality but did not show evidence for decreased accuracy or increased RT. The N2 amplitude was largest to incompatible trials but was reduced globally in the noise-induced condition with an overall attenuation in the N2 component. The fronto-central P3 was reduced as well to the incompatible trials for the noise induced group, whereas there were no differences found in the parietal Go-P3. Schapkin et al.

(2006a) suggested a physiological cost to those components contributing to the inhibition process (NoGo stimuli), whereas the components underlying overt responses (Go stimuli) were less vulnerable following noise induced sleep disturbance. This study illustrates the significance of poor sleep quality on inhibitory processes.

A follow-up study was conducted by the same group of researchers to investigate the impact of task difficulty on inhibitory functions following noise induced sleep disturbances (Schapkin, Falkenstein, Marks & Griefahn, 2006b). A very similar protocol was used as described earlier (Schapkin et al., 2006a) except participants were classified as 'good' verses 'poor' sleepers based on their subjective sleep quality scores. Also, two Go/NoGo tasks were used which varied in the level of difficulty. A total of 24 young adult participants with an equal gender mix completed the protocol. Schapkin et al. (2006b) went on to show that inhibitory processing was impaired in the poor sleep group as indexed by a reduction in P3 amplitude and an increase in P3 latency for NoGo trials in the difficult task only. The N2 amplitude was only marginally reduced in the poor sleepers after noise induction in the difficult task only. Although there were significant deficits for the poor sleep group following noisy nights, performance on accuracy and RT remained resilient to poor sleep quality. This study illustrates the significance of sleep quality on inhibitory processes. Individuals who report poorer sleep quality are more vulnerable to the effects of sleep loss on inhibitory functioning.

Breimhorst, Falkenstein, Marks and Griefahn (2008) adopted the same protocol as Schapkin et al., 2006a; Schapkin et al., 2006b except they analysed data for all participants after completing a Go/NoGo task in a well rested state. They then subjected all participants to noise throughout the night for the next few weeks and classified

participants as 'good' and 'poor' sleepers based on a sleep disturbance index. The N2 amplitude and the P3 latency to NoGo stimuli was reduced and delayed in those who reported poor sleep quality. In addition, the P3 to Go stimuli was reduced in the poor sleep group. Behavioural data was not different between groups. It was concluded that inter-individual differences pertaining to poor sleep quality affected the pre-motor inhibitory processes as indexed by the reduction in NoGo N2 amplitude, the speed of cognitive processing needed to stop an undesired response as indexed by the increase in NoGo P3 latency, and overall information processing to target information as indexed by the reduction on Go-P3 amplitude.

An fMRI study by Chuah, Venkatraman, Dinges and Chee (2006) showed individual differences in brain activation during a Go/NoGo task when sleep deprived. A total of 27 young adult mixed gender participants completed a 24 TSD protocol where they completed a Go/NoGo task in both a sleep deprived and well rested state. Sleep deprivation lowered activation in bilateral ventral and anterior prefrontal regions; however, those who were considered 'good performers' had right ventral PFC and right insula recruitment during the task. They also show increased activation in the ACC following false alarm hits. Chuah et al. (2006) postulated that after sleep deprivation, individuals might have difficulty activating ventrolateral PFC regions. Therefore, those who are resilient to sleep deprivation on a Go/NoGo task are better monitors as indexed by increased ACC activation, and must be able to recruit areas in the ventrolateral PFC and insular regions to be able to properly inhibit undesired responses.

Since changes in executive function and mood are typically robust findings in sleep deprivation literature, Anderson and Platten (2011) developed an experiment which

combined response inhibition and emotion regulation. They administered a Go/NoGo task which incorporated positive, negative and neutral words into a traditional response inhibition task. A sample of 32 young adult mixed gender participants were randomly assigned to either the control group or sleep deprivation group who remained awake for 36 hours. Results showed that sleep deprived individuals were more impulsive towards negative NoGo stimuli and produced faster RTs to negative NoGo's only. These data support the claim from the imaging work by Yoo et al. (2007) that reported a disconnect between the frontal lobe and the limbic system following sleep loss. Impulsivity towards negative stimuli may occur due to the impaired inhibitory control and disconnect between limbic areas.

Rationale and Hypothesis

This thesis project was part of a larger study designed to investigate the effects of sleep loss on frontal lobe function and emotion regulation. The focus of the thesis was strictly on performance monitoring using a letter Flanker and a response inhibition Go/NoGo task; emotion regulation data is not reported. EEG and ERPs were used to measure the electrophysiological brain response during task performance. Specific ERP components including stimulus-locked N2 and P3 as well as response-locked ERN and Pe were measured. These ERPs along with behavioural measures including response accuracy, RT and variability were examined to make observations about the effects of sleep deprivation on performance evaluation following response errors in situations with high response conflict.

This study aimed to add to the existing literature on performance monitoring during sleepiness in several ways. Performance monitoring was investigated in a large

sample (n = 49) using a between-subjects design to avoid problems with repeated-measures of performance tasks. Both stimulus-locked and response-locked ERPs were investigated in two different tasks, the oft-employed Flanker task, as well as a Go/NoGo (which has yet to be tested after total sleep deprivation) task to assess response inhibition. Using two tasks allowed for a more broad investigation of brain potentials associated with performance monitoring. Furthermore, the current study recorded EEG from 64 scalp sites to garner novel descriptive information on the dispersion of brain activity across the scalp during these tasks. An extreme level of sleep deprivation (28 hours) was investigated to fully challenge frontal lobe function in order to best test hypotheses about the influences of sleep loss on behaviour and brain function.

Based upon prior sleep deprivation research and especially Harrison and Horne's (2000) theoretical paper, it was expected that sleep deprivation would impact performance and brain physiology on these performance monitoring tasks. The prefrontal cortex comprises approximately 30% of the total cortical mass, yet it is the region with the greatest metabolic demand; thus Harrison and Horne (2000) suggest that the PFC is probably one of the first regions in the brain to become impacted during times of sleep deprivation due to its high demand of resources. This hypothesis is supported by the early fMRI work done by Drummond and colleagues (1999, 2000, 2001) showing the increased activation in additional PFC areas and increased activation (possibly compensation) in temporal and parietal areas. Given that the literature indicates performance monitoring (i.e., error monitoring and response inhibition) requires frontal top-down control, participants who perform a Flanker or Go/NoGo task after sleep loss should have difficulties in error, conflict resolution and response inhibition.

The current literature regarding performance monitoring and sleep deprivation remains inconclusive. A few studies have found behavioural differences (Tsai et al., 2005; Drummond et al. 2006; Hsieh et al., 2007; Hsieh et al., 2010; Anderson & Platten, 2011; Cain et al. 2011) on Flanker and response inhibition tasks, whereas others have not (Murphy et al., 2006; Schapkin et al., 2006a; Asaoka et al., 2010; Cain et al. 2011). Scheffers (1999), Murphy et al. (2006) and Tsai et al. (2005) have reported evidence for impaired remedial behaviour in sleep deprived individuals. The effect of sleep deprivation on stimulus and response locked ERP components also appear inconsistent across studies. Tsai et al. (2005) reported evidence for an enhanced N2 in sleep deprived participants; however, the majority of the literature suggests otherwise (Breimhorst et al., 2008; Schapkin et al., 2006a; Schapkin et al., 2006b). The literature regarding the NoGo-P3 and Go-P3 appear consistent such that the amplitude is reduced and latency delayed after sleep deprivation (Breimhorst et al., 2008; Schapkin et al., 2006a; Schapkin et al., 2006b). The majority of the sleep deprivation literature has found ERN differences (Scheffers et al., 1999; Tsai et al., 2005; Hsieh et al., 2007; Hsieh et al., 2010), but some studies have failed to find support for an effect of sleep deprivation; these studies used less extreme levels of sleepiness (Asaoka et al., 2010; Murphy et al., 2006). The Pe also appears attenuated when individuals are both sleepy (Asaoka et al., 2010; Murphy et al., 2006) and more extremely sleep deprived (Tsai et al., 2005; Hsieh et al., 2010), but has not been reported in all studies.

Based on these findings and the evidence that sleep deprivation impairs frontal lobe function, it was expected that when performing a Flanker and Go/NoGo task, sleep deprived participants would be less accurate, have slower RTs and respond more

variably. It was also expected that they would show deficits in their remedial behaviours such as post-error slowing and accuracy. It was expected that the N2 and P3 stimulus-locked ERP components and the ERN and Pe response-locked components would be attenuated in the sleep deprivation group compared to controls. These findings would show support for deficits in conflict and error detection as well as remedial strategy and response inhibition following sleep deprivation.

Method

Participants

Participants were recruited through the Brock University Psychology Department online recruitment system, classroom presentations and advertisements on campus bulletin boards, in campus newspapers and on social networking web sites (e.g., Facebook). To be considered an eligible candidate for participation, volunteers must have been between the ages of 18 and 30, healthy, good sleepers, right-handed, and free of any psychiatric conditions and traumatic brain injury. Successful study completion entailed a $110 honorarium or a $90 honorarium plus credit towards a university course.

A total of sixty-eight individuals met the inclusion criteria and were scheduled for participation in the experiment. Of the original sixty-eight, four were removed after polysomnography (PSG) screening for having either poor sleep efficiency (2) or periodic limb movements (2). Following PSG, seven individuals could not be scheduled for the experimental weekend. A total of eight participants were withdrawn during the experimental protocol due to: lack of interest (2), technical malfunctions (1), tolerance to sleep deprivation (2), personal scheduling conflicts (1) and poor electrophysiological

signal quality (2). Thus 49 participants completed the study. See Table 1 for a summary of participant demographics by group.

Materials

PSG nights were recorded using either Sandman (Tyco Inc, Ottawa) digital amplifiers and software or Neuroscan SynampsII amplifiers and software (Neuroscan, Inc., El Paso). Electrodes and sensors recorded electrocardiography (EKG), electromyography (EMG; anterior tibialis, submental), electrooculography (EOG; outer canthus of each eye), electroencephalography (EEG; O1, O2, C3 and C4), and chest and abdominal respiration. These recordings were used to identify breathing and movement disorders such as sleep apnea and periodic limb movement; these disorders affect daytime alertness and cognitive function. Impedances for PSG were maintained at 5 KΩ or less. EEG was referenced online to FPz and grounded at AFz with a sampling rate of 1000Hz and filtered 0.5 to 35Hz. Prior to sleep scoring, all EEG recordings were rereferenced offline to the contralateral mastoid site A1 or A2.

The protocol for the main study utilized similar sleep screening PSG procedures as the PSG protocol described above for baseline and experimental nights, with the exception of the respiration sensors and leg EMG. All experimental overnights were sampled at 1000 Hz, filtered 0.5 to 35Hz, and were recorded using Neuroscan SynampsII 64-channel amplifiers and SCAN v4.5 software (Neuroscan, Inc., El Paso). All electrode impedances for baseline and experimental sleep were maintained at 5 KΩ or less.

Wake electrophysiology was recorded using Neuroscan 64-channel Ag/AgCl Quikcaps. All waking EEG were referenced online to a central site in the cap (between Cz and CPz) and grounded at AFz. Prior to ERP analysis, all EEG were re-referenced

offline to the average of mastoid sites A1 and A2. Bipolar electrodes were used to record EKG (directly below each clavicle), EMG (submental chin), vertical and horizontal EOG (outer canthus of each eye). All waking electrode impedances were kept below 5 kΩ. Waking EEG was sampled at 1000 Hz and amplified using 64-channel digital SynampsII and Neuroscan software (Neuroscan, Inc., El Paso). Hardware filters used to record raw EEG were DC to 100Hz; which was later software filtered offline 1-20Hz FIR in response-locked ERPs and 1-30Hz FIR in stimulus-locked ERPs. Ear phones developed by Neuroscan were used to deliver auditory tones and a computer monitor was used to deliver visual stimuli using $STIM^2$ software (Neuroscan, Inc., El Paso).

Performance Assessment Battery

Participants completed the performance assessment battery (PAB) in one of three bedrooms assigned during the experimental protocol. The PAB was divided into two sections focusing on frontal lobe functioning and emotional processing respectively. Each section was approximately an hour and fifteen minutes in duration; the frontal lobe functioning PAB started at 10:30 the emotional processing PAB started at 14:00 Both sections of the PAB started with a Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, Smythe, Phillips and Dement, 1973) to evaluate subjective sleepiness and a positive and negative affect scale (PANAS; Watson, Clark and Tellegen, 1988). Refer to scales in Appendix A. Immediately following the paper and pencil subjective scales, an Alpha Attenuation Task (AAT) was used to measure physiological alertness and a six-minute auditory reaction time task to measure vigilance.

The frontal lobe function PAB included the Flanker (performance monitoring), Novel P3 (novelty processing), Go/NoGo (response inhibition) and the N-back (working

memory task). The emotional processing PAB included the International Affective Picture Set (IAPS) and face processing tasks. These tasks measured emotional processing to positive, negative and neutral pictures as well as happy, sad, angry and fearful facial expressions. See Table 2 and 3 for a complete chronological summary of each PAB.

The total duration of the Flanker task was fifteen minutes and was the first task following the AAT and RT tasks. Stimuli were presented using the letters "H" and "S" and were presented in grey on a black backdrop centered on the computer monitor. Participants were instructed to respond to the central target letter, which was flanked by an array of four other letters. Speed and accuracy were stressed equally to the participants. Congruent trials had flanking congruent letters that matched the central target whereas incongruent trials had a target letter flanked by incongruent letters. The Flanker task utilized 600 trials that were evenly divided into 4 blocks with breaks in-between. One third of the trials were congruent (HHHHH or SSSSS) and two thirds were incongruent (HHSHH, SSHSS). The stimulus duration time was 200 ms and had an inter-trial interval which varied randomly between 1200 and 1700 ms. Participants used a response pad that had the two target options labelled respectively and were instructed to use both hands, index fingers only. Response options were counterbalanced between bedrooms.

The Go/NoGo task was fifteen minutes in duration, and was administered following the Novel P3 and prior to the working memory task. Stimuli were presented using the symbols "X" and "+" and were presented in grey on a black backdrop centered on the computer monitor. Participants were instructed to respond to the target "X" and were to inhibit responses to the "+". Participants were encouraged to respond as quickly

as possible but to remain as accurate as possible. The Go/NoGo task contained 600 trials which were divided evenly into 4 blocks with breaks in-between. Eighty percent of the trials were the target Go "X" whereas the remaining twenty percent were NoGo "+" inhibition trials. The stimulus duration time was 50 ms and had an intertrial interval which varied randomly between 1000 and 2000 ms. Participants used the keyboard and were instructed to respond with the zero button on the numeric keypad and use the right index finger only.

Procedures

Individuals interested in participating in the study responded to one of the various types of recruitment strategies. Advertisements referred interested individuals to the Sleep Research Laboratory for a fifteen minute telephone interview. Questions about daily caffeine consumption, sleep/wake scheduling, medical conditions and body weight were used to verbally screen for healthy candidates. See Appendix B for telephone interview. If successful, those who screened as appropriate candidates were given an online access code to complete various questionnaires: Epworth Sleepiness Scale (Johns, 1991); trait version of the Stait-trait anxiety inventory, STAI-T (Spielberger, 1977); Yositake Fatigue scale (Yoshitake, 1978); Beck Depression Inventory (Beck, Ward, Mendleson, Mock, Erbaugh, 1961); Horne-Ostberg Morningness-Eveningness questionnaire (Horne, Ostberg, 1976); and, IPIP-Neo personality inventory (Goldberg and Saucier,1997).

Overnight PSG screening was scheduled after validation of the telephone interview and online questionnaires. Participants were to arrive to the laboratory at 21:00 on their scheduled PSG screening night. Upon arrival, participants were given a tour of

the laboratory facilities where they were shown the bedrooms, electrophysiological recording equipment, AV monitoring procedures and explained electrode hook up procedures. The research assistant then explained the study; participants were not to use any electronic devices (cell phones, laptops, and iPods) and outside food was not permitted. All meals were provided by the research assistants. If participants agreed to the study conditions, the research assistant then obtained signed consent. For consent letter see Appendix C. A hearing test was administered using a Welch Allyn Audiometer which was followed by overnight PSG electrode hook-up. Electrode placement adhered to the traditional 10-20 electrode placement system described by Pivik, Broughton, Coppola, Davidson, Fox and Nuwer (1993). Lights were turned off at 23:00 and participants were awakened at 7:00 the following morning which allowed each individual to sleep up to 8 hours. Prior to lights out and few minutes upon awakening, participants completed pre-and post-sleep questionnaires that were developed by the Brock University Sleep Research Laboratory. For pre- and post-sleep questionnaires see Appendix D. After electrodes were removed, participants were given the opportunity for a shower and breakfast. Before leaving participants were asked to complete sleep diaries with an online access code a week prior to their scheduled study weekend. PSG records were evaluated for presence of sleep disordered breathing and periodic limb movements.

44 Hour in-Laboratory Protocol

The total duration of the laboratory protocol was 44 hours; participants were randomly assigned to either a control group where they obtained a full night rest or a sleep deprivation group where they remained awake for 34 hours. See Figure 1 for a

summary of the pre-study and 44-hour in laboratory protocol. The experimental protocols ran from Thursday evenings to Saturday evenings.

The arrival times for participants were 21:00 and 21:30 on baseline night (Thursday) and were 21:00 and 21:30 on the experimental night (Friday). All electronic devices and outside food were temporarily removed and electronics turned off. Participants were then asked on the baseline night to complete emotion questionnaires: Behavioural Inhibition Scale and Behavioural Activation Scale; BIS-BAS (Carver and White, 1994); Emotion Regulation Questionnaire; ERQ (Gross and John, 2003); Barratt Impulsiveness Scale version 11; BIS-11 (Patton, Stanford and Barratt, 1995); and, if female, menstrual cycle questionnaire. A practice PAB was then administered to familiarize participants with all the tasks used in the experimental PAB on both nights. Research assistants verified tasks were understood by checking accuracy of data. If not enough errors were committed on Flanker and Go/NoGo tasks, it was repeated with the instruction to increase the speed of responses. Similar baseline and experimental PSG electrode placement was used as the PSG screening night. Prior to lights out at 23:00, and after lights on at 07:00, participants were asked to complete pre- and post-sleep questionnaires and provide a saliva samples for the purpose of recording sleep quality and circadian endocrine function. Before leaving the laboratory Friday morning all participants were required to sign a consent agreeing to avoid: napping, caffeine, nicotine, alcohol, exercise, medication, and to engage in proper eating behaviours.

After completion of a second practice PAB on the experimental night, participants were notified of their experimental condition. If assigned to the sleep deprivation condition, all participants remained awake with two research assistants for the duration of

the night. Participants were asked to complete a pre-sleep questionnaire and provide a saliva sample at 22:40 to record baseline circadian effects in testosterone, estradiol and cortisol. Participants were explicitly told that they could expect to feel very sleepy but it is important to remain motivated to stay awake. Research assistants and participants engaged in board games, card games and movies. Calorie restricted snacks were given at 1:00, 3:00 and 5:00. PANAS, SSS and visual analog mood scales were administered every hour starting at 23:00 and finishing the next day at 14:00 to record subjective sleepiness and mood regulation. At 7:00 a post-sleep questionnaire was completed and participants were asked to again provide a saliva sample. Both groups were given the opportunity for a shower and were provided with breakfast.

Electrode application for the frontal lobe functioning PAB started at 9:15 Saturday morning. The first PAB concluded at 12:00 with the NASA Task Load index which indexed mental, physical and temporal demands, as well as subjective performance, effort and frustration (Hart & Staveland, 1988). Lunch was provided by research assistants which included submarine sandwiches with the choice of diet decaffeinated soda or water. The second emotion regulation PAB started at 14:00 with a PANAS, SSS and state version of the Stait-trait anxiety inventory, STAI-S (Spielberger, 1977). The second PAB concluded at 15:30 with a second NASA Task Load index. Upon PAB completion participants had approximately 30 minutes to clean up and relax prior to the Point Subtraction Aggression Paradigm (PSAP). At 16:00 participants provided a saliva sample and were guided though the PSAP instructions where they were told they would be competing in an online game of competition where they could increase their honorarium depending on their performance. The study concluded at 16:30. Participants

received a study feedback form from the research assistants (see Appendix F) and were cautioned (if in the sleep deprivation group) about the effects of excessive sleepiness. Research assistants ensured all participants had a way of transportation home.

Data Analysis

To ensure each participant in the control group obtained an adequate amount of sleep on the experimental night, sleep efficiency was analyzed by dividing the total time asleep by the total time in bed. A trained sleep technician scored all sleep electrophysiology according to Rechtschaffen and Kales (1968) sleep scoring procedures.

The Flanker and Go/NoGo stimulus-locked ERPs were epoched from -100ms to 900ms and baseline corrected from -100ms to zero. Prior to epoching the stimulus-locked trials, EEG cleaning was conducted to remove any noisy signals from the raw EEG recordings. Manual artifact rejection was done first by visually inspecting each raw EEG recording for any bad electrode channels for artifact (e.g., 60 Hz noise, EEG clipping, EMG artifact). After data cleaning, eye regression was conducted for each participant using Neuroscan software. Automatic artifact rejection consisted of Neuroscan software removing additional artifact on all channels where the VEOG channel exceeded ±100 μV. Each epoch was then inspected manually on each channel for all stimuli to remove trials on which there was any unwanted electrophysiological signals. Individual averages for correct responses were then computed by stimulus type. Individual averages were FIR and bandpass filtered at 1-30Hz (6db/octave) for stimulus-locked, and 1-20Hz (6db/octave) for response-locked ERPs. Grand averages were then computed, for each group.

The Flanker and Go/NoGo response-locked ERPs were analyzed using the same procedures as the stimulus-locked ERPs except that all response-locked ERPs were epoched from -800ms to 600ms. The baseline used for the response-locked ERPs utilized a sweep correction range of -800 ms to -600ms. By drawing a baseline behind stimulus onset, this method avoided stimulus effects which have been shown to sometimes appear in response-locked ERP averages (Verleger, Japkowski, & Wascher, 2005; Murphy et al., 2006). In the Flanker task, individual averages for error responses were calculated by combining all error trials regardless of congruency. The rationale for developing a composite error ERP with both congruent and incongruent error trials was because too few errors were committed on congruent trials and preliminary analyses showed no differences in ERPs across trial type (Christ, Falkenstein, Heuer, Hohnsbein, 2000). In the Go/NoGo task, individual averages for error responses were calculated by averaging all the incorrectly inhibited NoGo trials.

A mean amplitude peak detection method was used to measure the Pe component generated to correct and error trials in Flanker and Go/NoGo tasks as clearly defined peaks are not often observed. Nieuwenhuis et al. (2001) choice of a 200 to 400 ms range was adopted for the Flanker task, and a 50 to 400 ms range was adopted for the Go/NoGo task as this was when the Pe began and finished its peak in the grand average waveform. Although the entire 64-channel montage was investigated, electrode sites FCz and Pz are reported herein as these were the sites where both stimulus-locked and response-locked ERPs were largest.

Due to the confounding nature of lapses in NoGo accuracy in the Go/NoGo task after sleep deprivation, criteria were set for determining the validity of a correct NoGo

inhibition. Specifically, if a lapse in attention were to occur surrounding a NoGo trial, the failure to respond would appear correct inhibition. Thus, for a NoGo trial to be considered valid, participants must have responded correctly to the preceding and following Go trials surrounding the NoGo trial. See Figure 2 for an illustration.

Since the sleep deprived participants were expected to respond significantly slower, RT variability was calculated by using the coefficient of variation (a ratio of the standard deviation divided by the mean RT) because of expected group differences in the magnitude of mean RT. For independent samples t-test and mixed-model analysis of variance (ANOVA) procedures, Levene's test was used to determine homogeneity of variance and Mauchly's test was used to determine violations to sphericity. Any violations were corrected using the corrected values (e.g., Greenhouse-Geisser for ANOVA). All distributions were explored for outliers (e.g., ±3 standard deviations) and tested for normality using Kolmorgorov-Smirnov test. Trends are discussed where outcomes were in the expected direction of the hypotheses. All statistical analyses were conducted using SPSS statistical software. Significance levels were set at $p \leq .05$.

Results

Validation of Sleep Deprivation Manipulation

Sleep Architecture. In order to verify that groups had comparable sleep on the baseline night, sleep architecture was compared between groups. Independent t-tests showed that the sleep deprivation and control group did not differ on sleep architecture variables on Baseline night (all ps >.05). See Table 4. Sleep efficiency (SE) was 93.07% (*SD* = 5.12) for control and 93.40% (*SD* = 4.20) for sleep deprivation group on Baseline night; control participants obtained an overall sleep efficiency of 95.00% (*SD* = 3.00) on

the Experimental night. Further, comparisons were made between Baseline and the Experimental night in controls to verify they slept well on both nights. Compared to Baseline, controls spent less time in Stage 1 $t(23) = 2.289, p = .032$ and more time in SWS $t(23) = -2.584, p = .017$, and REM $t(23) = -2.860, p = .009$ on the Experimental night. See Table 5.

Deficits in RT, Subjective Sleepiness and Mood. In order to confirm that sleep deprivation leads to deficits in RT, subjective sleepiness, and mood consistent with prior research, groups were compared on typical measures including: performance on a simple RT task at 10:30 and 14:00, ratings on fatigue, SSS, and VAS mood taken pre-and post-sleep on two nights in laboratory, and repeated measures of SSS and PANAS taken at regular intervals throughout the duration of the study. See Table 6 thorough 10 for means and standard deviations.

Group (Control, Sleep Deprivation) by Time of Day (AM, PM) mixed-model ANOVAs were conducted to investigate mean RT, coefficient of RT variation, mean 10% fastest, inverse of mean 10% slowest, missed trials and lapse trials (i.e., RTs greater than 500ms). A Time of Day main effect was found for mean RT, $F(1, 44) = 5.96, p = .019, \eta^2 = .12$ mean 10% slowest, $F(1, 44) = 6.88, p = .012, \eta^2 = .14$, and lapse trials, $F(1, 44) = 4.68, p = .036, \eta^2 = .10$. Collectively, participants mean RTs were slower, their 10% slowest RTs were slower and they lapsed more in the afternoon compared to the morning trials. Group main effects were found for mean RT, $F(1, 44) = 29.4, p < .001, \eta^2 = .40$, coefficient of RT variation, $F(1, 44) = 54.4, p < .001, \eta^2 = .55$, mean 10% fastest, $F(1, 44) = 12.2, p = .001, \eta^2 = .22$, mean 10% slowest, $F(1, 44) = 42.3, p < .001, \eta^2 = .49$, missed trials, $F(1, 44) = 19.1, p < .001, \eta^2 = .30$, and lapses, $F(1, 44) = 30.0, p < .001, \eta^2 =$

.41. The sleep deprived group had a slower mean RT, larger coefficient of RT variation, slower mean 10% fastest and 10% slowest RTs, more missed trials, and more lapses, collapsed across Time of Day. No interactions were found for any of the RT variables. For descriptive statistics regarding RT variables see Table 6.

Subjective sleepiness, fatigue and VAS mood scales were surveyed in pre-and post-sleep questionnaires administered on Thursday 22:40, Friday 07:00, Friday 22:40, and Saturday 07:00. Group (Control, Sleep Deprivation) by Time (Thursday 22:40, Friday 07:00, Friday 22:40, Saturday 07:00) mixed-model ANOVAs were run to investigate the effects of TSD. There was a significant Group by Time interaction for subjective sleepiness, $F(3, 129) = 8.74, p < .001, \eta^2 = .17$, fatigue, $F(3, 141) = 10.7, p < .001, \eta^2 = .19$, and for the VAS categories calm/irritable, $F(3, 129) = 5.36, p = .004, \eta^2 = .11$, energetic/sluggish, $F(3, 129) = 2.94, p = .036, \eta^2 = .06$, and relaxed/tense $F(3, 129) = 3.99, p = .018, \eta^2 = .09$. After remaining awake all night (Saturday 07:00), in comparison to the well rested controls, the sleep deprived group reported more subjective sleepiness, $t(46) = -3.94, p < .001$, fatigue, $t(47) = -3.71, p = .001$, irritability, $t(33) = -3.47, p = .001$, sluggishness, $t(47) = -2.87, p = .006$, and tenseness, $t(31) = -2.79, p = .009$. A Group, $F(1, 44) = 5.79, p = .020, \eta^2 = .12$, and Time, $F(3, 132) = 4.61, p = .009, \eta^2 = .10$, main effect was found for the happy/sad VAS scale, but no interaction. Nevertheless, an independent sample t-test was conducted that showed the sleep deprived group reported more sadness at 07:00 Saturday morning in comparison to the controls on the happy/sad VAS mood scale $t(47) = -2.32, p = .025$. Groups did not differ on any baseline measures (Thursday 22:40, Friday 07:00, Friday 23:40; p's >.05) except for fatigue, $t(47) = 3.24, p = .002$ on Friday 23:40. This group difference is likely due to the

fact that participants knew group assignment at the time they completed pre-sleep questionnaire on Friday evening. See Table 7 through 9 for descriptive statistics.

The SSS and PANAS was used to track subjective sleepiness and positive and negative affect across the 44 hour in laboratory protocol. Independent sample t-tests showed no statistical differences in subjective sleepiness on the baseline night (Thursday), baseline morning (Friday) or experimental night (Friday; all p's>.05). Various independent samples t-tests across the experimental day showed that the sleep deprived group reported more subjective sleepiness across all time intervals (all p's $\leq$ .001). See Table 9 for descriptive statistics and Figure 3 for a visual representation. As well, independent samples t-tests showed group differences in subjective positive affect on PANAS across all time intervals (Saturday 08:00-16:00); the sleep deprived group had significantly lower positive mood (all p's <.001). Similarly, greater subjective negative affect was reported in the sleep deprived group compared to the controls from 08:00 to 14:00 Saturday (all p's $\leq$ .05). See Table 10 for descriptive statistics and all t-tests. See Figure 4 for a visual representation of PANAS positive and Figure 5 for PANAS negative mood.

Error Monitoring Using a Flanker Task

Behavioural Data. Group (control, Sleep Deprivation) by Stimulus Type (congruent, incongruent) mixed-model ANOVAs were conducted to assess behavioural accuracy, coefficient of RT variation and omission rate across groups. For response accuracy, a Stimulus Type main effect was found, $F(1, 48) = 141.8$, $p < .001$, $\eta^2 = .75$, but a Group effect and Stimulus Type by Group interaction were not evident. All participants made more errors to incongruent trials ($M = 15.14\%$, $SD = 7.92$) in comparison to

congruent trials (M = 6.61%, SD = 5.16). For coefficient of RT variation, a Group main effect was found, $F(1, 48) = 7.69$, $p = .008$, $\eta^2 = .14$, but a Stimulus Type effect and Stimulus Type by Group interaction were not evident. The sleep deprived group (M = 0.24, SD = .04) was more variable than controls (M = 0.21, SD = .04) collapsed across congruent and incongruent trials. For omission rate, a main effect of Stimulus Type was found, $F(1, 48) = 5.69$, $p = .021$, whereas a Group effect and Stimulus Type by Group interaction were not evident. All participants missed more incongruent trials (M = 6.23%, SD = 6.50) compared to congruent trials (M = 5.51, SD = 5.81).

To assess behavioural RT, a Group (Control, Sleep Deprivation) by Stimulus Type (congruent, incongruent) by Accuracy (correct, error) mixed-model ANOVA was conducted. A significant Stimulus Type by Accuracy interaction was found, $F(1, 48)$ = 31.8, p <.001, $\eta^2 = .40$. When participants responded correctly, they were significantly slower to incongruent trials (M = 404.51ms, SD = 44.46) compared to congruent trials (M = 372.24ms, SD = 42.34); $t(49) = -14.0$, p <.001. RT to incorrect trials did not differ significantly across trial type. Main effects for Stimulus Type, $F(1, 48) = 16.5$, p <.001, $\eta^2 = .26$, Accuracy, $F(1, 48) = 150.1$, p <.001, $\eta^2 = .76$, and Group, $F(1, 48) = 6.16$, p = .017, $\eta^2 = .11$, were also observed. Sleep deprived (M = 375.24 ms, SD = 41.69) participants responded significantly slower than controls (M = 345.95 ms, SD = 42.55) collapsed across Stimulus Type and Accuracy. See Figure 6 for a visual representation of RT differences between trial types. Collectively, these behavioural measures indicate the presence of the Flanker "congruency effect". Participants made more errors, had increased omissions and responded significantly slower to incongruent trials compared to

congruent trials due to the increased conflict and reduced cognitive control (van Veen and Carter, 2002a). See Table 11 for all Flanker behavioural data.

Post-error Remedial Behaviour. Previous research has shown that individuals typically slow down following an incorrect response; this is an adaptive mechanism that introduces corrective behaviours by increasing the likelihood of responding correctly on the following trial (Notebaert et al., 2009). To test this, a Group (Control, Sleep Deprivation) by Stimulus Type (correct, error) mixed model ANOVA was run to investigate remedial slowing post error responses. Reaction times were taken on trials following a correct response as well as trials following error responses and submitted to the 2 by 2 ANOVA. A main effect of Group was found, $F(1, 48) = 5.98$, $p = .018$, $\eta 2 = .11$, but there was no evidence for a Stimulus Type main effect nor a Stimulus Type by Group interaction. The sleep deprived group ($M = 408.92$ms, $SD = 41.21$) was significantly slower than controls ($M = 380.39$ms, $SD = 41.21$) on both correct responses following a correct response and correct responses following an error response collapsed. See Table 12 for means and standard deviations. An independent samples t-test was also run to compare groups on remedial accuracy following an incorrect response. A trend was found suggesting that the sleep deprived group ($M = 86.58\%$, $SD = 8.95$) was less accurate than controls ($M = 90.87\%$, $SD = 8.57$) on trials following an incorrect response, $t(48) = 1.73$, $p = .09$.

Stimulus-locked ERPs

N2. Group (Control, Sleep Deprivation) by Stimulus type (Congruent, Incongruent) mixed-model ANOVAs were run to investigate the differences in N2 amplitude and latency at electrode site FCz. For N2 amplitude, a Stimulus Type main

effect was significant, $F(1, 47) = 8.87$, $p = .005$, $\eta^2 = .16$, but there was no Group main effect or interaction. Collectively, all participants generated larger N2 amplitudes to incongruent trials ($M = -5.03$ μV, $SD = 2.44$) compared to congruent trials ($M = -4.53$ μV, $SD = 2.54$). For N2 latency, there was a significant main effect of Stimulus Type, $F(1, 46) = 10.45$, $p = .002$, $\eta^2 = .19$, but there was no Group main effect or interaction. The N2 was earlier to congruent trials ($M = 267.76$ms, $SD = 28.62$) compared to incongruent trials ($M = 273.71$ms, $SD = 14.80$) for all participants. See Table 13 for N2 amplitude and latency means and standard deviations.

P300. Stimulus Type (Congruent, Incongruent) by Group (control, sleep deprivation) mixed-model ANOVAs were run to investigate P3 amplitude and latency at both FCz and Pz electrode sites. For P300 amplitude at FCz, there were no main effects or interaction. For P300 latency at FCz, there was a significant main effect of Stimulus Type, $F(1, 46) = 23.7$, $p < .001$, $\eta^2 = .34$, but no main effect of Group or interaction. P300 was delayed for incongruent ($M = 382.98$ms, $SD = 31.73$) compared to congruent trials ($M = 362.75$ms, $SD = 28.28$) at FCz for both groups collapsed. For P300 amplitude at Pz, there were no main effects or interaction. For P300 latency at Pz, there was a significant main effect of Stimulus Type, $F(1, 47) = 5.42$, $p = .024$, $\eta^2 = .10$, and Group, $F(1, 47) = 13.14$, $p = .001$, $\eta^2 = .22$, but no interaction. The P300 was delayed for incongruent trials ($M = 373.86$ms, $SD = 54.92$) compared to congruent trials ($M = 355.86$ms, $SD = 44.81$) at Pz. The sleep deprived group ($M = 383.92$ms, $SD = 37.48$) had a significantly delayed P300 in comparison to the controls ($M = 344.29$ms, $SD = 38.26$) to both the congruent trials and incongruent trials combined. See Table 13 for P300 means and standard deviations. See Figures 7 through 10 for stimulus-locked Flanker ERPs.

Response-locked ERPs

ERN. A paired samples t-test showed error trials produced significantly larger ERNs (M = -6.32μV, SD = 3.83) at the FCz frontal electrode site compared to trials which participants responded correctly (M = 1.70 μV, SD = 2.76), $t(48) = 15.0$, $p < .001$. Contrary to expectation, no significant difference in ERN amplitude was observed between controls (M = -7.07 μV, SD = 4.24) and sleep deprived participants (M = -5.59 μV, SD = 3.32); $t(47) = -1.36$, $p = .180$. Pearson correlations were computed to assess the relationship between ERN amplitude and performance on the Flanker task. There was a negative correlation between incongruent accuracy and ERN amplitude for both controls (r = -.55, n = 24, $p = .005$) and sleep deprived participants (r = -.46, n = 25, $p = .021$). See Figure 11 for scatter plots. This moderately strong negative correlation suggests those who make more errors on the Flanker task (as indexed by high incongruent error rates) have smaller ERN amplitudes. To further explore the mechanism underlying the relationship between ERN amplitude and error rate, a correlation analysis was run between the coefficient of RT variation to incongruent correct trials, incongruent accuracy and ERN amplitude. The coefficient of RT variation was negatively correlated with incongruent accuracy in controls (r = -.68, n = 24, $p < .001$) and (r = -.61, n = 26, p = .001) in sleep deprived individuals, whereas the coefficient of RT variation to incongruent correct trials was positively correlated with ERN amplitude in the controls only (r = .53, n = 24, $p = .007$). See Figure 12 and 13 for the respective scatter plots.

Given the relationship between ERN amplitude and incongruent accuracy and since sleep deprived participants were expected to systematically make more errors, follow up analyses were run to investigate the hypothesized effect of sleep deprivation in

the ERN for both groups with a comparable number of trials in the ERP. Specifically, a subsample (n = 20 per group) of participants were investigated who made at least 20+ errors on the Flanker task. The first 20 artifact free error responses for each participant were included to compare groups on ERN amplitude. In this analysis that controlled for the number of trials, the ERN was significantly smaller in the sleep deprived group compared to the controls as expected, $t(38) = -2.19, p = .035$. See Table 14 for means and standard deviations. See Figure 14 and 15 for ERP waveforms.

Due to the finding that controlling for an equivalent number of trials changed the outcome of the ERN amplitude, another follow up analysis was conducted to explore changes in ERN response over time. The sample was further reduced to a subgroup (n = 9 for controls and 11 for sleep deprivation) of participants who made at least 40+ errors. A Group (control, Sleep Deprivation) by Error Block (First 20 trials, Last 20 trials) mixed model ANOVA was run to investigate changes in ERN amplitude across time on the Flanker task. There was a significant Group by Error Block interaction, $F(1, 18) = 5.063$, $p = .037$, $\eta^2 = .22$. Follow-up paired samples t-tests showed that the sleep deprived group did not differ in ERN amplitude between the first 20 artifact free errors and last 20 artifact free errors, whereas a trend was found in the controls suggesting the ERN reduces in amplitude (possibly because of habituation) with an increase in error rate over time, $t(8) = -2.22, p = .057$. See Table 15 for means and standard deviations and Figure 16 for the interaction.

An independent samples t-test showed no significant difference in ERN latency measured at FCz between controls ($M = 67.96$ ms, $SD = 15.56$) and sleep deprivation ($M = 70.76$ ms, $SD = 18.66$), $t(47) = -0.57, p = .570$. Pearson correlations were computed to

assess relationships between ERN latency and performance on the Flanker task. No significant correlations were observed between any of the performance measures and ERN latency (all p's>.05).

Pe. A paired samples t-test showed error trials (M = 3.00μV, SD = 3.20) produced significantly larger mean Pe amplitudes at the Pz electrode site compared to trials which participants responded to correctly (M = -3.09μV, SD = 2.01), $t(48) = 11.8$, $p < .001$. An independent samples t-test showed no significant difference in Pe mean amplitude between controls (M = 2.74μV, SD = 3.04) and sleep deprived participants (M = 3.23μV, SD = 3.38); $t(47) = -.527$, $p = .601$. Pearson correlations were computed to assess any relationships between Pe mean amplitude and performance on the Flanker task. There was a positive correlation between incongruent accuracy and Pe mean amplitude for controls ($r = .46$, n = 24, $p = .022$) and a trend in sleep deprived participants ($r = .39$, n = 25, $p = .052$). See Figure 17 for scatter plots. This moderate positive correlation suggests those who perform worse on the Flanker task (as indexed by high incongruent errors) have a smaller mean Pe amplitude.

Since accuracy impacted Pe mean amplitude, a subsample of participants were investigated if they made more than 20+ errors on the Flanker task. The first 20 artifact free error responses were then subjected to an independent samples t-test to further investigate Pe mean amplitude across groups. No significant difference in Pe mean amplitude was observed. See Table 14 for means and standard deviations. See Figure 14 and 15 for ERP waveforms. Due to the finding that controlling for an equivalent number of trials changed the outcome of the ERN electrophysiology and there was a correlation between error rate and Pe amplitude, an exploratory follow up analyses was conducted to

explore changes in Pe mean amplitude over time. The sample was further reduced to the subgroups (n = 9 for controls and 11 for sleep deprivation) of participants who made more than 40+ errors. A Group (Control, Sleep Deprivation) by Error Block (First 20/Last 20) mixed model ANOVA was run to investigate changes in Pe amplitude across time on the Flanker task. There was a significant main effect of Group, $F(1, 18) = 6.00$, $p = .025$, $\eta^2 = .25$, but no effect of Error block or interaction. When error trials were collapsed across error block, the sleep deprived group had significantly larger Pe mean amplitudes. See Table 15 for means and standard deviations.

Response Inhibition Using a Go/NoGo Task

Behavioural Data. Group (Control, Sleep Deprivation) by Stimulus Type (Go, NoGo) mixed-model ANOVAs were run to investigate the differences in response accuracy and RT. For response accuracy, a main effect of Stimulus Type, $F(1, 47) = 283.3$, $p < .001$, $\eta^2 = .86$, and Group, $F(1, 47) = 8.91$, $p = .004$, $\eta^2 = .16$, was found but no interaction. All participants made more errors to NoGo ($M = 43.95\%$, $SD = 16.56$) compared to Go stimuli ($M = 6.8\%$, $SD = 7.72$), and the sleep deprived group ($M = 29.6\%$, $SD = 10.0$) made more errors than controls ($M = 21.3\%$, $SD = 9.50$) collapsed across both trial types. Although there was no interaction, given the fundamental differences in stimuli type, exploratory t-tests were run on both Go and NoGo trials. Significant differences were observed between groups on Go trials, but not on NoGo trials. For RT, a main effect of Stimulus Type, $F(1, 47) = 293.7$, $p < .001$, $\eta^2 = .86$, but no effect of Group or interaction was found. All participants responded faster to unsuccessful NoGo inhibitions ($M = 299.04$ ms, $SD = 29.83$) compared to properly

executed Go Stimuli (M = 337.56 ms, SD = 35.10). See Table16 for t-tests, means and standard deviations for all Go/NoGo behavioural data.

Stimulus-locked ERPs

NoGo-N2. Stimulus Type (Go, NoGo) by Group (Control, Sleep Deprivation) mixed-model ANOVAs were run to investigate the differences in N2 amplitude and latency across groups. A Group by Stimulus Type interaction, $F(1, 43) = 11.5$, $p = .002$, $\eta^2 = .21$, was found for N2 amplitude. A trend was found suggesting that the sleep deprived group produced a smaller NoGo-N2 compared to the control group, $t(43) = -1.79$, $p = .080$, whereas groups did not differ on Go-N2 amplitude. A main effect of Stimulus Type, $F(1, 43) = 7.25$, $p = .010$, $\eta^2 = .14$, but no effect of Group or interaction was found regarding N2 latency. All participants experienced delayed NoGo-N2 (M = 282.82 ms, SD = 29.29) compared to Go-N2 (M = 272.78 ms, SD = 27.45) ERPs. See Table 17 for means and standard deviations. See Figure 18 for NoGo-N2 ERP waveforms and Figure 19 for NoGo ERP topography.

Go-P3. The Go-P3 ERP was measured at electrode site Pz; it was investigated using independent samples t-tests comparing controls and sleep deprived participants on amplitude and latency. Controls had significantly larger Go-P3 amplitudes but did not differ in latency compared to the sleep deprived group. See Table 17 for means, standard deviations and t-tests. See Figure 20 for Go-P3 ERP waveforms and Figure 21 for ERP topography.

NoGo-P3. The NoGo-P3 physiology was measured at electrode site FCz; it was investigated using independent samples t-tests comparing controls and sleep deprived participants on amplitude and latency. Controls and sleep deprived did not differ

significantly on amplitude or latency. See Table 17 for means, standard deviations and t-tests. See Figure 18 for NoGo-P3 ERP waveforms and Figure 19 for NoGo ERP topography.

Response-locked ERPs

ERN. Group (Control, Sleep Deprivation) by Stimulus Type (Correct, Error) mixed-model ANOVAs were run to investigate the differences in ERN amplitude and latency at FCz. A Group by Stimulus Type interaction was found, $F(1, 42) = 12.93$, $p = .001$, $\eta^2 = .24$, for ERN amplitude. The sleep deprived participants showed significantly smaller ERN amplitudes to unsuccessful NoGo inhibitions compared to the control group, $t(42) = -3.53$, $p = .001$, but groups did not differ in correct-related negativity amplitude. See Table 18 for means and standard deviations. A main effect of Stimulus Type was found for ERN latency $F(1, 42) = 6.97$, $p = .012$, $\eta^2 = .14$, but no effect of Group or interaction. Collectively, the ERN to correct trials ($M = 28.86$ ms, $SD = 19.78$) was significantly later compared to the ERN to error trials ($M = 26.20$ ms, $SD = 20.78$). See Figure 22 for Go/NoGo response-locked ERP waveforms and Figure 23 for Go/NoGo response-locked topography. Pearson correlations were computed to assess any relationships between ERN amplitude and objective performance on the Go/NoGo task. No significant relationships were observed between Go/NoGo performance and ERN amplitude.

Pe. A Group (Control, Sleep Deprivation) by Stimulus type (Correct, Error) mixed-model ANOVA was run to investigate the differences in Pe mean amplitude at Pz. A main effect of Stimulus Type was found for Pe mean amplitude, $F(1, 42) = 156.0$, $p < .001$, $\eta^2 = .79$ but no effect of Group or interaction. Collectively, all participants

produced larger Pe amplitudes to error trials (M = 3.72μV, SD = 2.44) compared to correct trials (M = -1.48μV, SD = 1.21). See Table 19 for Pe mean amplitude means and standard deviations. Pearson correlations were computed to assess any relationships between Pe mean amplitude and objective performance on the Go/NoGo task. No significant relationships were observed between Go/NoGo performance and Pe mean amplitude.

Discussion

A total sleep deprivation protocol was run to investigate individuals' behaviour and underlying electrophysiological correlates of performance monitoring and response inhibition. The well-rested controls obtained a sleep efficiency on the experimental night of 95%; therefore, they were an appropriate comparison group. The sleep deprived participants reported more subjective sleepiness and impaired mood states and had slowed RT, more variability, and more lapses on a simple RT task compared to controls. These data confirm that the sleep deprivation manipulation led to robust effects on constructs that previous literature has reliably shown to be susceptible to TSD. Sleep deprived individuals also responded slower on the Flanker task and tended to be less accurate on trials following incorrect responses suggesting a minor impairment in their remedial behaviour. Although no group differences were observed in false alarm rate, sleep deprived participants had a lower accuracy in Go hit rate on the Go/NoGo task. The performance monitoring system was impaired as indexed by reductions in ERN on both tasks and NoGo-N2 ERPs. The stimulus-locked P3 components were found to be delayed in the Flanker task and the amplitude reduced to Go-trials in the Go/NoGo task. Correlations between performance accuracy and ERN amplitude on the Flanker task

showed that ERN amplitude attenuated in the well rested group as error rate increased, whereas ERN remained constant in the sleep deprived group irrespective of changes in error rate. The Pe was larger in the sleep deprived group compared to the control group for a sub group who performed relatively poorly on the Flanker task. ERPs were recorded from multiple sites in order to describe any changes in topography (e.g., hemisphere differences, anteriorization/posteriorization of potentials, increases reflecting compensation). ERP components of interest (e.g., N2, P3, ERN, Pe) were maximal where expected and effects were fairly widespread across the scalp; there did not appear to be interactions between sleep deprivation and topography.

Error-monitoring

Based on previous evidence that sleep deprivation impairs frontal lobe functioning, it was expected that Flanker performance would be susceptible to sleep deprivation such that participants would be less accurate, have slower RTs, respond more variably and show deficits in their remedial behaviours. Along with the behavioural measures, the stimulus-locked and response-locked ERP components were expected to be delayed and attenuated. This study showed that sleep deprivation led to deficits in RT and RT variability which is consistent with previous research (Scheffers et al., 1999; Tsai et al., 2005; Hsieh et al., 2009; Hsieh et al., 2010), but failed to show accuracy differences reported previously. As the sleep deprived participants also had delayed P3 ERP waveforms to Flanker stimuli, the slow RT may be due to deficits in stimulus evaluation (Donchin and Coles, 1988) as a result of sleep deprivation. Despite previous literature reporting impairments in remedial behaviour, post-error slowing and accuracy (Scheffers et al., 1999; Tsai et al. 2005; Murphy et al., 2006), this study did not find evidence for a

slowing effect, but did find a trend in post-error accuracy suggesting sleep deprived participants were less accurate on trials following an error.

The response-locked ERP effects are currently mixed in the error monitoring and sleep deprivation literature. Correlation analyses showed an inverse relationship between the ERN amplitude and incongruent error rate in both well-rested and sleep deprived groups. Specifically, the larger the error rate, the smaller the ERN. This relationship was first observed by Gehring et al. (1993) who reported that the ERN was smallest when speed was stressed over accuracy, unchanged when speed and accuracy were equally stressing, and largest when accuracy was stressed over speed. This relationship was also observed by Hajcak et al. (2003) and Hermann et al. (2004) such that participants who made fewer errors produced larger ERNs.

A response control hypothesis was developed by Pailing, Segalowitz, Dywan, and Davies (2002) that purported that individuals with larger ERNs were expected to have smaller error rates and smaller response RT differences reflecting a more controlled response strategy. Pailing et al. (2002) did not find evidence for a significant ERN/error rate correlation but did report a relationship between RT differences and ERN suggesting that those with less of a RT difference between correct and error trials produced larger ERNs. Although this hypothesis appears plausible, Hajcak et al. (2003) noted that the relationship between the ERN and error rate could also represent a 'habituation' response to making errors. The data reported in this thesis lend support for this habituation effect. Specifically, data illustrate that ERN amplitude changed as a function of error block as indicated by the interaction observed between the first and last 20 error blocks in a subsample of participants who made more than 40+ errors. The ERN for well-rested

controls was smaller in the last error block (last 20 error trials) compared to the first error block (first 20 trials). This effect was not found in the sleep deprived group suggesting that the ERN remains constant (i.e., habituation intact in well rested controls but disrupted after sleep deprivation) despite changes in error rate in the sleep deprived group. This habituation interpretation is supported by Holroyd and Coles' (2002) reinforcement learning dopamine hypothesis. If error signals are fed back to the ACC via the dopamine system to modify performance, and there is no consequence with repeated errors, then habituation may occur leading to reduced activity in the ACC. This result illustrates the importance of selecting not only an equivalent number of trials, but also an early set of trials to draw ERN waveforms because habituation may reduce the ERN effect in well-rested controls.

Gosselin, De Koninck and Campbell (2005) showed using a novelty processing task that sleep deprived participants showed attenuated frontal novel P3 waveforms with a more posterior parietal shift. They concluded that compensatory mechanisms must be used after TSD because novel stimuli require additional parietal processing (as indexed by a second P550 component). An alternate explanation may be that the TSD participants are treating novel stimuli like targets. Where the rested participants quickly decipher the difference between novel and target stimuli, the sleep deprived individuals may require additional processing and lack the ability to habituate to novel stimuli. Further research should be carried out to investigate the absence of habituation after sleep deprivation in tasks that require frontal lobe function.

Murphy et al. (2006) and Asaoka et al. (2010) used prolonged wakefulness (20 hours awake; a four-hour bedtime delay) and sleep inertia protocols respectively. Both

protocols subjected participants to a "sleepy" state and yielded results conflicting with those of the current study and others (Scheffers et al., 1999; Hsieh et al., 2007). Specifically, both Murphy et al. (2006) and Asaoka et al. (2010) reported no evidence for differences in ERN amplitude but found reduced Pe amplitudes in their sleepy participants. The current study, as well as many others (Scheffers et al., 1999; Tsai et al., 2005; Hsieh et al., 2007; Hsieh et al., 2010), found evidence for reduced ERN amplitudes following TSD. It may be that the frontal neural physiology remains unaffected or mildly affected in subtle levels of sleepiness produced by four hours of prolonged wakefulness or sleep inertia. That the Pe component appears affected by these subtle degrees of sleepiness may be because mood and affective state are altered and thus emotional evaluation to errors is impaired (Asaoka et al., 2010). The fact that ERNs were smaller for the sleep deprived group in the current study supports the hypothesis for sleep deprivation leading to a deficit in the frontal regions of the brain (Harrison and Horne, 2000); both the ERN and NoGo-N2 have been shown to be generated in the dorsal ACC (van Veen and Carter, 2002b). Thus, the dorsal ACC may not be impaired during minor sleep delays or sleep inertia. In contrast, ventral affective areas of the ACC may be more susceptible during subtle levels of sleepiness. Balkin et al. (2002) has reported that the functional connectivity between the ACC and other brain regions is stable at 5 and 20 minutes post-awakening response (a time of high sleep inertia). This result may explain why ERP components like the ERN are less affected by sleep inertia and small amounts of prolonged wakefulness (Asaoka et al., 2010).

The Pe mean amplitude was larger in the sleep deprived group compared to controls for participants who made more than 40+ errors on the Flanker task. In poor

Flanker performers, it may be that where controls habituate to making mistakes (smaller ERN over time with increased error rate), the sleep deprived individuals may perseverate on their mistakes. This hypothesis can be supported by the recent imaging research showing the disconnect between the PFC and limbic areas in sleep deprived individuals (Yoo et al., 2007). It is important for future research to investigate the effects of varying degrees of sleepiness on ACC function (specifically ventral areas) as this is the area of Pe generation (van Veen and Carter, 2002b).

The null results in ERN amplitude reported by Murphy et al. (2006) and Asaoka et al. (2010) may possibly be attributed to the number of trials they included in their ERP averages. The current study illustrates the importance of controlling for an equal number of trials in the ERP individual averages as the ERN amplitude attenuates in well-rested individuals who perform poorly on the Flanker task (poorer performance correlated with smaller ERN amplitude; thus more trials in the average would wash out the sleep deprivation effect).

Response Inhibition

It was expected that response inhibition performance would be susceptible to sleep deprivation such that participants would be less accurate, have slower RTs, and respond more impulsively to NoGo stimuli. The stimulus-locked Go-P3, NoGo-N2 and NoGo-P3 as well as response-locked Go/NoGo ERN and Pe ERP components were expected to be delayed and attenuated. The current study is the first to investigate response-locked electrophysiology to a Go/NoGo response inhibition task after TSD. All of the current literature used noise-induced sleep disturbance protocols (Breimhorst et al., 2008; Schapkin et al., 2006a; Schapkin et al., 2006b) or were behavioural studies

(Drummond et al., 2006). Congruent with the Flanker literature, reduced ERN amplitudes were observed to false-positive NoGo stimuli in the sleep deprived group. These data support the hypothesis that sleep deprived individuals have impaired error detection as indexed by the attenuation in ERN amplitude.

The more commonly studied stimulus-locked ERPs showed reductions in the NoGo-N2, which is consistent with Breimhorst et al. (2008), Schapkin et al. (2006a), and Schapkin et al. (2006b). Tsai et al. (2005) did not find support for reduced N2 amplitude in their sleep deprived group, which may be due to task differences. The reduced NoGo-N2 amplitudes observed in this study and others have been induced by high conflicting NoGo stimuli, whereas the Tsai et al. (2005) study, an arrow Flanker task was used. The reduction in NoGo-N2 amplitude in the sleep deprived group thus reflects impairment in the inhibitory network (probably at a pre-motor level; Breimhorst et al., 2008).

The deficit in Go-P3 amplitude reported here is also consistent with Breimhorst et al. 2008, Schapkin et al. 2006a, and Schapkin et al. 2006b. These authors also found evidence for delayed NoGo-P3 latencies, which was not found in the current study. Gosselin et al. (2005) reported attenuated P300 amplitudes in sleep deprived individuals who underwent a novel processing task. Collectively, this study and others suggest a deficit in the resources necessary for normal levels of information processing after sleep deprivation.

Drummond et al. (2006) showed that after 23 hours of TSD, individuals increased in false negative rate, and at 55 hours, decreased their hit rate. The current study supports the decreased hit rate after approximately 30 hours of TSD, but failed to support the difference in false positive rate. However, Breimhorst et al. (2008), Schapkin et al.

(2006a), and Schapkin et al. (2006b), also reported no behavioural differences on their response inhibition tasks. Past imagining studies showed automatic responding to targets activates areas in the middle frontal gyrus (Yamasaki, LaBar, & McCarthy, 2002) and the superior frontal sulcus (Culham, Cavanagh, & Kanwisher, 2001). Deficits in these frontal regions may explain the deficits in sustained attention and poor hit performance in the sleep deprived participants (Drummond et al., 2006).

An alternative explanation for these behavioural differences may be due to task difficulty. The well-rested controls obtained an accuracy of 60% to NoGo trials; these data suggest extreme difficulty compared to the response inhibition task used in Drummond et al. (2006) study. The highest degree of behavioural impairment in Drummond et al. (2006) study occurred at 31 hours TSD with an overall accuracy of 80% (false positive rate of only 20%). The Drummond et al. (2006) task utilized NoGo shapes that had similar perceptual features as the Go stimuli. The current study used 'X' as Go and '+' as NoGo stimuli which may have introduced some degree of cognitive interference. According to Harrison and Horne (2000), difficult tasks boost motivation and compensation in sleep deprived individuals. Therefore, brain areas that are involved in motor inhibition may have been compensated for due to the high task difficulty.

No relationships were observed in the Go/NoGo task with respect to behavioural and electrophysiological measures. This finding may have been due to an equivalent number of trials for each participant in the Go/NoGo grand averages; whereas the Flanker had a more unequal number of trials per participant. See Appendix G and H for number of trials. Thus, the inter-subject variability of trials initially subjected to the ERN generation for the Flanker was larger than the inter-subject variability for the Go/NoGo.

Applications

Recent societal changes have led to increased pressure to produce resources for a rapidly growing population. This demand necessitates cycling shift work for 24-hour production. Sustained attention during night shifts is very important as many disasters have occurred during early morning hours; fatigue and sleepiness have been suggested as key contributors to such disasters (Dinges et al., 1989). Professionals who specialize in medicine, transportation or security must be able to excel during times of high conflict monitoring. These professionals must be able to innovate and solve problems; both of these qualities require adequate levels of frontal lobe function. Taffinder, McManus, Gul, Russell, and Darzi (1998) showed that medical surgeons who remained awake all night made more errors, spent more time on tasks, experienced more stress and had reduced arousal levels compared to well-rested residents. Since TSD decreases cognitive function (attention, vigilance and memory) and mood/motivation in medical residents after only one night of being 'on-call', patient health and safety may be at risk (Lingenfelser et al., 1994).

The impact of sleep deprivation on frontal lobe function and performance monitoring may be especially relevant for adolescents and older adult age groups because of the age-related compromised frontal lobe function. Past literature has shown adolescence to be a period of development marked by poorly defined frontal function with adolescents demonstrating poorer inhibitory control compared to younger adults (Luna & Sweeney, 2004). Research has shown that adolescents are also chronically sleep deprived (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998). Adolescents have different circadian cycles causing them to stay awake later due to phase delayed

melatonin secretions. Early school start times (causing reduced school-night sleep) have been associated with daytime sleepiness (Carskadon et al., 1998), depressive mood, and performance deficits in adolescents (Wolfson, & Carskadon, 1998). Similarly, previous research has reported performance monitoring deficits in older adults (Dywan, Mathewson and Segalowitz, 2004). Older adults reported more errors, slower RTs and attenuated ERN and Pe components on a letter Flanker task. Major sleep changes are also reported in the aging population such that their sleep is lighter (more stage 1 and 2 relative to slow wave and rapid eye movement) and more fragmented (increased arousals; Bliwise, 2011). Developmental changes along with excessive daytime sleepiness caused by reduced or fragmented sleep, may lead to compounding deficits in frontal brain function for vulnerable populations like adolescences and the elderly; further research is needed in these groups to determine if sleep deprivation leads to greater impairment in performance monitoring.

Limitations

A limitation to the current study is the very narrow range for the age of the participants. Although groups were well balanced for age and gender, the overall age range for the participants was 18 to 24. This narrow age range is a potential issue as past literature has suggested younger individuals (20-22 years of age) are more resilient to sleep deprivation compared to older adults (40-44 years of age) on tasks that involve visual search, reasoning and vigilance (Webb & Levy, 1982). Contrary to this, Phillips et al. (2004) showed that 20-25 year olds' RT increased as sleep deprivation increased, whereas 52-63 year olds' RT remained unchanged on a simple RT task across time. This result may be explained by effects of motivation and conscientiousness or floor effects in

older adults. Nonetheless, it is important to investigate the potential differences in performance monitoring after sleep deprivation across the lifespan as the current data cannot be generalized to the wider population.

Task differences have been suggested as a potential reason for the inconsistent results regarding sleep deprivation and performance monitoring (Harrison & Horne, 2000). Harrison and Horne (2000) suggested that highly difficult tasks produce weaker sleep deprivation effects. They argue that the more complex and "rule-based" the task, the greater the motivation and compensation a sleep deprived individual will expend. This has been observed in an imaging study by Chee and Choo (2004) who showed behavioural performance changed as a function of task complexity after sleep deprivation. They also showed increased frontal activation during a complex task and interpreted this as a compensation strategy to overcome the effects of sleep deprivation. An EEG study showed that when sleep deprived participants responded correctly on a Flanker task, they did so with significantly more beta activity at the beginning of the trial compared to well rested individuals (Hsieh et al., 2009). This beta activation suggests that the sleep deprived participants were more motivated or provided more effort when responding correctly because beta EEG indexes cognitive engagement (Prinzel et al., 2000; Lorenzo et al., 1995; Corsi-Cabrera et al., 1996). The current study used a letter Flanker and Go/NoGo task that are considered very difficult tasks. Controls responded correctly to incongruent stimuli in the Flanker with an accuracy of 85%; whereas, they responded correctly to NoGo stimuli only 59% of the time. The observed NoGo accuracy for well-rested controls is very low compared to other literature (85% in Drummond et al., 2006; 88% in Anderson & Platten, 2011). This accuracy discrepancy suggests our

Go/NoGo task was very difficult compared to other literature. According to Harrison and Horne (2000), some of the null effects observed in the behavioural measures may be due to the sleep deprived group increasing compensatory effort due to the complexity of tasks or controls becoming unmotivated because of task difficulty.

Future Directions

Future research is needed to determine changes in the underlying neural physiology during performance monitoring during periods of sleep inertia (i.e., upon awakening), short-term prolonged wakefulness (i.e., 18-20 hours), cumulative sleep restriction (e.g., reducing sleep to 5 hours over consecutive nights), total sleep deprivation (i.e., greater than 24 hours), and after recovery sleep. A dose-response sleep deprivation study or repeated measures design may resolve some of the lingering issues between the aforementioned studies. Further, introducing multiple types of Flanker and Go/NoGo tasks is also a potential avenue for future research. Since task complexity appears to be a potential issue in sleep deprivation research, many Flanker (arrow, letter, global/local) and response inhibition (Go/NoGo, Stroop) tasks exist that could address these issues. The intertrial interval and stimulus duration could also be manipulated to increase or decrease task difficulty level. Further research is also needed to address the issue of error rate and ERP habituation/response control on tasks used for performance monitoring research. The current study showed the importance of equal trials in individual ERP averages. It would also be of interest to investigate individual differences in performance monitoring behaviour and ERP physiology to understand the nature of performance variables and factors associated with vulnerability or resiliency to sleep deprivation.

Conclusions

The current sleep deprivation protocol provides evidence for compromised performance monitoring in a very large sample of individuals as indexed by behavioural data and decreases in ERN, NoGo-N2 and Go-P3 amplitudes found in Flanker and Go/NoGo tasks. Well-rested individuals habituated with increased error rate (supported by a reduction in ERN amplitude as error rate increased), whereas sleep deprived individuals remained in a stable state throughout the task regardless of error rate. Go-P3 amplitudes were reduced after sleep deprivation and NoGo-N2 tended to be smaller suggesting deficits in context updating and conflict detection. These data add to a body of evidence showing that the frontal brain region is particularly vulnerable to sleep loss following both the traditional Flanker and newly tested Go/NoGo task. Although topography differences were not reported, it is a future avenue for researchers interested in the possible brain compensation following sleep deprivation. Contributing to the understanding of the neural basis of these deficits in performance monitoring abilities is particularly important for our increasingly sleep deprived society and for safety and productivity in situations like driving and the workplace.

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Table 1

Summary of Participant Demographics by Age and Group

	Control		Sleep Deprivation		Total	
	N	Mean age	N	Mean age	N	Mean age
Males	13	19.23	11	20.55	24	19.83
Females	12	19.25	13	19.15	25	19.20
Total	25	19.24	24	19.79	49	19.51

Table 2

Summary of the Frontal Lobe Functioning Performance Assessment Battery

Task	Measure	Duration (minutes)
SSS and VAS Mood	Subjective Sleepiness and Mood	1
PANAS	Positive and Negative Affect	2
Alpha Attenuation	Physiological Alertness	4
Simple Reaction Time	Psychomotor Vigilance	6
Flanker	Error Processing	15
Break	Break	10
Novel P3	Novelty Processing	15
Go/No-Go	Response Inhibition	15
2-back Memory	Working Memory	10
NASA Effort	Effort and Motivation Scale	2

Table 3

Summary of the Emotional Processing Performance Assessment Battery

Task	Measure	Duration (minutes)
SSS and VAS Mood	Subjective Sleepiness and Mood	1
PANAS	Positive and Negative Affect	2
STAI-state	State Anxiety Scale	1
Alpha Attenuation	Physiological Alertness	4
Simple Reaction Time	Psychomotor Vigilance	6
IAPS Emotion	Emotional Processing	10
Break	Break	10
Full Intensity - Face Processing	Emotional Processing	15
Morphed - Face Processing	Emotional Processing	25
NASA Effort	Effort and Motivation Scale	2

Table 4

Sleep Architecture on Baseline Night in Sleep Deprivation and Control Groups

	Control		Sleep Deprivation				
	M	*SD*	*M*	*SD*	*df*	*t*	*p*
Minutes							
Wake	22.66	24.11	20.69	20.27	46	0.152	.880
Stage 1	57.45	35.24	45.94	19.12	35	1.406	.168
Stage 2	196.27	38.98	204.59	23.27	38	-0.898	.375
SWS	121.42	36.37	113.01	29.20	46	0.883	.382
REM	71.59	26.12	83.83	21.89	46	-1.759	.085
Movement	11.58	5.02	10.87	4.88	46	0.495	.623
Total sleep time	446.73	24.93	447.36	22.29	46	-0.093	.927
Time in bed	479.98	2.10	478.93	6.88	27	0.715	.481
Onset to Stage 1	13.17	17.24	15.69	19.39	46	-0.476	.636
Onset to Stage 2	20.90	18.24	25.83	19.62	46	-0.902	.372
Consolidated Stage 2 SO	44.12	33.38	36.14	32.14	46	0.844	.403
Onset to REM	150.98	56.23	140.39	50.57	46	0.686	.496
Percent							
Wake	4.52	5.02	4.33	4.24	46	0.136	.893
Stage 1	11.98	7.37	9.59	3.97	35	1.398	.171
Stage 2	40.89	8.13	42.72	4.85	37	-0.945	.350
SWS	25.28	7.51	23.59	6.05	46	0.861	.394
REM	14.91	5.43	17.50	4.53	46	-1.789	.080
Movement	2.41	1.05	2.27	1.01	46	0.489	.627
Sleep Efficiency	93.07	5.12	93.40	4.20	46	-0.242	.810

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 24 for both groups.

Table 5

Sleep Architecture on Baseline and Experimental Nights for Control Group

	Baseline		Experimental				
	M	*SD*	*M*	*SD*	*df*	*t*	*p*
Minutes							
Wake	21.66	24.11	13.25	12.68	23	1.503	0.146
Stage 1	57.45	35.24	43.48	20.36	23	2.289	**0.032**
Stage 2	196.27	38.98	191.25	39.34	23	0.726	0.475
SWS	121.42	36.37	135.25	31.08	23	-2.584	**0.017**
REM	71.59	26.12	87.26	23.80	23	-2.860	**0.009**
Movement	11.58	5.02	11.39	3.06	23	0.210	0.835
Total sleep time	446.73	24.93	457.24	12.80	23	-1.862	0.075
Time in Bed	479.98	2.11	481.88	3.38	23	-3.425	**0.002**
Onset to Stage 1	13.17	17.24	8.69	10.20	23	1.101	0.282
Onset to Stage 2	20.90	18.24	13.29	10.62	20	2.189	**0.041**
Consolidated Stage 2 SO	44.12	33.38	45.88	35.27	23	-0.179	0.860
Onset to REM	150.98	56.23	128.44	51.82	23	1.695	0.104
Percent							
Wake	4.52	5.02	2.75	2.64	23	1.513	0.144
Stage 1	11.98	7.37	9.03	4.26	23	2.316	**0.030**
Stage 2	40.89	8.13	39.67	8.07	23	0.849	0.405
SWS	25.28	7.51	28.07	6.47	23	-2.517	**0.019**
REM	14.91	5.43	18.11	4.94	23	-2.791	**0.010**
Movement	2.41	1.05	2.36	0.64	23	0.263	0.795
Sleep Efficiency	93.07	5.12	94.89	2.54	23	-1.576	0.129

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 24 for all conditions except for onset to Stage 2, N = 21 due to the removal of 3 outliers.

Table 6

Reaction Time (RT) Data During the AM and PM Performance Assessment Batteries

	Control		Sleep Deprivation	
Time	*M*	*SD*	*M*	*SD*
10:30 AM				
Mean RT	307.74	36.28	404.04	89.30
Coefficient of RT variability	.17	.04	.26	.08
Mean 10% Fast	236.63	29.15	289.36	68.47
Mean 10% Slow	418.37	66.46	647.85	161.63
Missed Trials	.68	1.04	8.4	12.68
Number of Lapses	.91	1.41	8.29	8.56
2:00 PM				
Mean RT	325.53	43.59	426.48	81.14
Coefficient of RT variability	.18	.05	.30	.08
Mean 10% Fast	247.09	27.58	277.60	47.62
Mean 10% Slow	449.96	98.32	715.73	157.77
Missed Trials	.73	1.04	11.17	9.84
Number of Lapses	1.68	3.29	11.04	6.83

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 22 for control and 24 for sleep deprivation.

Table 7

Descriptive Statistics for the Fatigue Scale Between Groups on Baseline and Experimental Conditions

		Control			Sleep Deprivation		
Time	Condition	*N*	*M*	*SD*	*N*	*M*	*SD*
Thursday 22:40	Baseline Pre-sleep	25	3.48	1.42	24	3.25	1.39
Friday 07:00	Baseline Post-sleep	25	2.80	1.12	24	2.75	1.22
Friday 22:40	Experimental Pre-sleep	25	3.28	.98	24	2.86	1.02
Saturday 07:00	Experimental Post-sleep	25	3.00	1.26	24	4.50	1.59

Note: *M* = means; *SD* = standard deviation; Fatigue scale on 7-points where higher number reflects greater fatigue.

Table 8

Descriptive Statistics to the Visual Analog Scale (VAS) for mood Between Groups Baseline and Experimental Days

		Control			Sleep Deprivation		
Time	Condition	*N*	*M*	*SD*	*N*	*M*	*SD*
Calm/Irritable							
Thursday 22:40	Baseline Pre-sleep	22	13.00	11.27	23	17.87	13.33
Friday 07:00	Baseline Post-sleep	22	15.86	9.99	23	20.00	19.76
Friday 22:40	Experimental Pre-sleep	22	11.41	8.66	23	19.87	16.37
Saturday 07:00	Experimental Post-sleep	22	13.32	11.86	23	35.57	26.96
Happy/Sad							
Thursday 22:40	Baseline Pre-sleep	23	14.43	12.21	23	19.35	14.97
Friday 07:00	Baseline Post-sleep	23	19.52	14.41	23	24.00	16.99
Friday 22:40	Experimental Pre-sleep	23	15.43	13.57	23	22.39	15.55
Saturday 07:00	Experimental Post-sleep	23	18.65	15.54	23	34.48	23.35
Energetic/Sluggish							
Thursday 22:40	Baseline Pre-sleep	22	45.68	23.89	23	51.35	23.58
Friday 07:00	Baseline Post-sleep	22	34.95	16.55	23	40.09	25.29
Friday 22:40	Experimental Pre-sleep	22	41.32	19.40	23	41.22	20.62
Saturday 07:00	Experimental Post-sleep	22	41.18	21.87	23	64.26	26.23
Relaxed/Tense							
Thursday 22:40	Baseline Pre-sleep	22	17.27	18.76	23	18.26	16.01
Friday 07:00	Baseline Post-sleep	22	15.50	12.59	23	17.61	13.06
Friday 22:40	Experimental Pre-sleep	22	11.95	11.55	23	17.35	14.67
Saturday 07:00	Experimental Post-sleep	22	13.86	13.01	23	31.35	28.03

Note: *M* = means; *SD* = standard deviation; higher number reflects greater irritability, sadness, sluggishness and tenseness.

Table 9

Participants Subjective Sleepiness Measured by the Stanford Sleepiness Scale (SSS)

		Control			Sleep Deprivation					
		N	*M*	*SD*	N	*M*	*SD*	*df*	*t*	*p*
Thursday 22:40	Baseline Pre-sleep	23	3.13	1.42	24	2.96	1.30	46	.423	.674
Friday 7:00	Baseline Post-sleep	24	2.61	.94	23	2.61	.99	44	.000	1.000
Friday 22:40	Exp. Pre-sleep	25	2.84	.94	23	2.39	.78	46	1.784	.081
Saturday 7:00	Exp. Post-sleep	25	2.64	.95	23	4.09	1.50	46	-3.942	< .001
Saturday 8:00	25 hours awake	25	1.60	.65	24	3.25	1.36	47	-5.391	< .001
Saturday 9:00	26 hours awake	25	1.56	.82	24	3.54	1.35	47	-6.176	< .001
Saturday 10:30	27.5 hours awake	25	1.96	.89	24	4.83	1.27	47	-9.121	< .001
Saturday 12:00	29 hours awake	23	2.35	1.30	24	4.25	1.42	45	-4.779	< .001
Saturday 13:00	30 hours awake	25	1.96	.79	24	2.96	1.08	47	-3.699	.001
Saturday 14:00	31 hours awake	25	2.16	1.07	24	4.17	1.58	47	-5.190	< .001
Saturday 16:00	33 hours awake	25	1.40	.65	24	2.54	1.02	47	-4.658	< .001

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; Exp. = Experimental. Baseline Post-sleep is considered time zero where experimental pre-sleep is 16 hours awake for both groups. Experimental post-sleep is zero hours awake for controls and 24 hours awake for sleep deprived individuals. Higher number reflects greater subjective sleepiness.

Table 10

Positive and Negative Affect Measured by PANAS

		Control			Sleep Deprivation					
Time of day	Hours awake	*N*	*M*	*SD*	*N*	*M*	*SD*	*df*	*t*	*p*
Positive										
8:00	25	25	32.36	7.67	24	20.83	9.12	47	4.797	< .001
9:00	26	25	32.24	7.74	24	19.21	9.00	47	5.440	< .001
10:30	27.5	25	29.96	8.97	24	15.46	6.09	47	6.595	< .001
12:00	29	23	25.26	8.04	24	15.04	5.72	45	5.004	< .001
13:00	30	25	28.24	6.61	24	18.79	7.93	47	4.538	< .001
14:00	31	25	27.12	7.98	24	15.79	6.76	47	5.352	< .001
16:00	33	23	34.17	7.12	24	22.58	10.41	45	4.419	< .001
Negative										
8:00	25	23	10.43	.95	24	11.46	1.53	45	-2.77	.009
9:00	26	23	10.65	1.03	24	11.67	2.08	45	-2.14	.040
10:30	27.5	23	10.86	1.46	24	12.42	2.55	45	-2.57	.014
12:00	29	21	11.62	1.72	24	14.88	4.05	43	-3.59	.001
13:00	30	23	10.65	1.11	24	11.83	2.50	45	-2.11	.043
14:00	31	23	10.96	1.61	24	11.67	1.90	45	-1.38	.173
16:00	33	21	10.76	1.14	24	11.95	2.82	43	-1.91	.066

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom. Higher number reflects greater mood.

Table 11

The Effects of Flanker Congruency and Sleep Deprivation on Accuracy, Omission Rate and Reaction time

	Control				Sleep Deprivation			
	Congruent		Incongruent		Congruent		Incongruent	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Accuracy (%)	94.55	5.01	86.17	6.97	92.33	5.01	83.65	8.67
Omissions (%)	4.17	4.90	4.95	6.13	6.75	6.38	7.41	6.71
RT Correct (ms)	355.71	35.93	385.20	36.86	387.50	42.68	347.08	63.33
RT Error (ms)	325.59	63.74	317.29	36.40	422.34	44.00	344.02	46.40
Co. RT variation	.21	.03	.21	.04	.24	.04	.24	.06

Note: *M* = means; *SD* = standard deviation; Co. = coefficient; N = 24, for control and N = 26 for sleep deprived group.

Table 12

Remedial Reaction Times (ms) to Correct Trials following Correct and Error Responses

	Correct Response			Incorrect Response		
	N	*M*	*SD*	N	*M*	*SD*
Control	24	379.05	37.40	24	381.72	40.43
Sleep Deprivation	26	406.76	45.26	26	411.07	49.99
Total	50	393.46	43.56	50	396.98	47.55

Note: *M* = mean; *SD* = standard deviation; N = sample size.

Table 13

Effects of Flanker Congruency and Sleep Deprivation on Stimulus-locked ERPs

	Control				Sleep Deprivation			
	Congruent		Incongruent		Congruent		Incongruent	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
N2 at FCz								
Amplitude (μV)	-4.33	2.77	-4.90	2.66	-4.74	2.30	-5.16	2.20
Latency (ms)	260.38	18.06	270.33	13.61	268.83	18.17	276.71	14.80
P300 at FCz								
Amplitude (μV)	7.80	3.35	7.78	2.94	6.58	2.70	6.21	2.81
Latency (ms)	358.63	25.80	374.17	25.23	366.88	30.55	391.79	35.47
P300 at Pz								
Amplitude (μV)	9.50	3.14	9.58	2.95	8.80	2.82	8.13	2.87
Latency (ms)	341.46	33.60	347.13	59.77	369.68	50.27	398.16	35.78

Note: *M* = means; *SD* = standard deviation; N = 24 for controls and 25 for SD. N = 24 for the SD group on the latency variable for N2 and P300 at FCz due to the removal of one outlier.

Table 14

Flanker Error-related Negativity and Error-Positivity to the First 20 Artifact Free Incorrect Responses Compared Between Groups in a sub sample of Individuals who Made 20+ errors.

	Control		Sleep Deprivation	
	M	*SD*	*M*	*SD*
ERN				
First 20	-7.78	4.24	-5.18	3.22
Pe				
First 20	2.67	2.91	2.46	3.26

Note: *M* = means; *SD* = standard deviation; N = 20 for both groups.

Table 15

Flanker Error-related Negativity and Error-Positivity to the First 20 and Last 20 Artifact Free Incorrect Responses Compared Between Groups in a sub sample of Individuals who Made 40+ Errors.

	Control		Sleep Deprivation	
	M	*SD*	*M*	*SD*
ERN				
First 20	-6.31	4.92	-4.79	2.72
Last 20	-4.32	4.39	-5.05	2.74
Pe				
First 20	0.25	1.43	1.58	2.12
Last 20	0.14	1.60	2.41	1.83

Note: *M* = means; *SD* = standard deviation; N = 9 for controls and N = 11 for SD.

Table 16

The Effects of Response Inhibition and Sleep Deprivation using a Go/NoGo Task on Accuracy, and Reaction time

	Control		Sleep Deprivation				
	M	*SD*	*M*	*SD*	*df*	*t*	*p*
Accuracy (%)							
Go	97.52	2.33	88.71	8.80	47	4.749	<.001
NoGo	59.80	15.57	52.15	16.96	47	1.645	.107
RT (ms)							
Go	331.85	31.56	343.51	38.21	47	-1.167	.249
NoGo	291.73	29.55	306.66	28.76	47	-1.792	.080

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 25 for controls and 24 for SD.

Table 17

The Effects of Response Inhibition and Sleep Deprivation using a Go/NoGo Task on Stimulus-locked ERPs

	Control		Sleep Deprivation				
	M	*SD*	*M*	*SD*	*df*	*t*	*p*
NoGo-N2							
Amplitude (μV)	-6.46	3.39	-4.79	2.82	43	-1.792	.080
Latency (ms)	277.91	26.72	287.95	31.55	43	-1.154	.255
NoGo -P3							
Amplitude (μV)	11.75	3.90	11.14	4.36	43	.496	.622
Latency (ms)	391.09	32.12	410.00	42.87	43	-1.680	.100
Go- P3							
Amplitude (μV)	6.84	2.62	5.15	2.80	42	2.069	.045
Latency (ms)	295.83	32.43	312.45	32.29	41	-1.680	.101

Note: *M* = means; *SD* = standard deviation; *df* = degrees of freedom; N = 23 for controls and 22 for sleep deprived, except for Go-P3; a sleep deprived participant was removed on latency because it was an outlier and another was removed due to a poor Pz recording channel. NoGo-N2 and NoGo-P3 electrophysiology was measured at FCz whereas Go-P3 was measured at Pz.

Table 18

Error-related Negativity to Correct and Incorrect Responses by Group at FCz for Go/NoGo task

	ERN Amplitude				ERN Latency			
	Correct		Incorrect		Correct		Incorrect	
	M	*SD*	*M*	*SD*	M	*SD*	*M*	*SD*
Control	2.31	2.55	-9.15	3.15	31.00	19.20	28.32	19.68
Sleep Deprivation	1.49	2.47	-5.72	3.29	26.73	20.57	24.09	22.09
Total	1.90	2.52	-7.44	3.63	28.86	19.78	26.20	20.78

Note: *M* = mean; *SD* = standard deviation. N = 22 per group

Table 19

Error Positivity to Correct and Incorrect Responses by Group at Pz for Go/NoGo task

	Pe Amplitude			
	Correct		Incorrect	
	M	*SD*	*M*	*SD*
Control	-1.84	1.03	3.37	2.62
Sleep Deprivation	-1.12	1.30	4.08	2.26
Total	-1.48	1.21	3.72	2.44

Note: *M* = mean; *SD* = standard deviation. N = 22 per group.

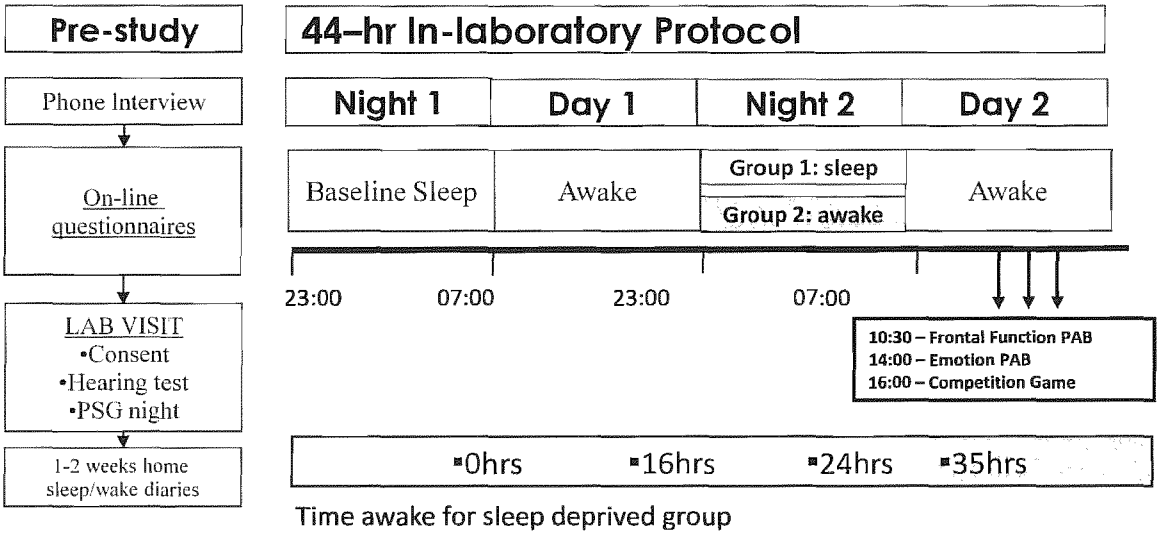


Figure 1. Pre-study and 44 hour in-laboratory protocol. The pre-study protocol includes phone interview and PSG lab visit. The 44 hour in lab protocol includes Night 1, Day 1, Night 2 and Day 2. A timeline for the total time awake for the SD group is provided at the bottom.

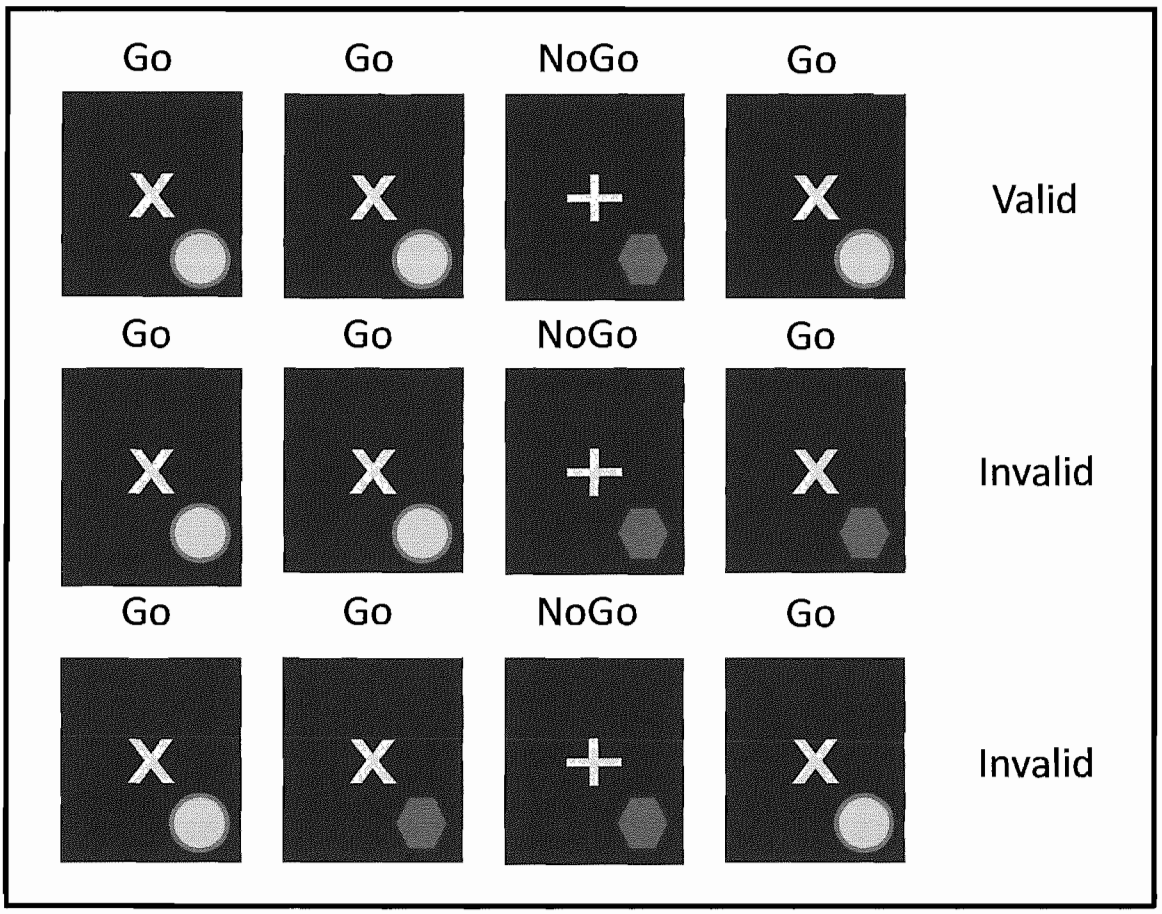


Figure 2. Criteria for classifying NoGo trials as valid inhibitions. For a NoGo trial to be considered valid, participants must have responded correctly to the preceding and following Go trial surrounding the NoGo trial. Otherwise lapses on NoGo trials could have been erroneously considered successful inhibitions. In the illustration above, the "X" represents a Go trial, whereas a "+" represents a NoGo trial. Circles represent an accurate hit, octagons represent a missed response, which could have been either a lapse (on Go trials) or inhibition (on NoGo trials).

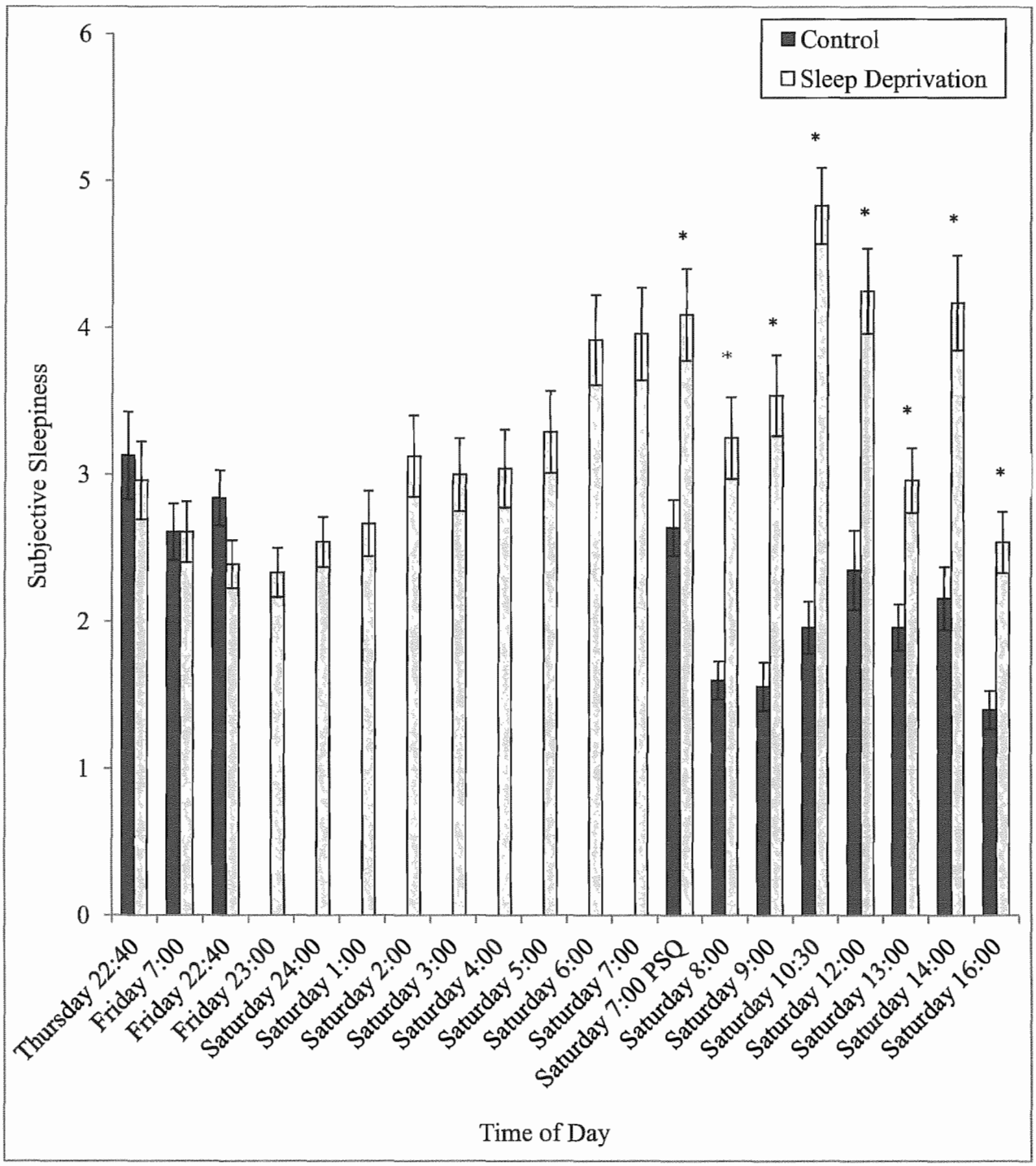


Figure 3. Subjective sleepiness measured by the Stanford Sleepiness Scale (SSS). No initial differences were found on Baseline night (Thursday 22:40), Baseline morning (Friday 7:00), or the Experimental night pre-sleep (Friday 22:40). The asterisks* represent significant differences in subjective sleepiness and error bars represent standard error of the mean. Note the liner increase in subjective sleepiness overnight in the sleep deprived group.

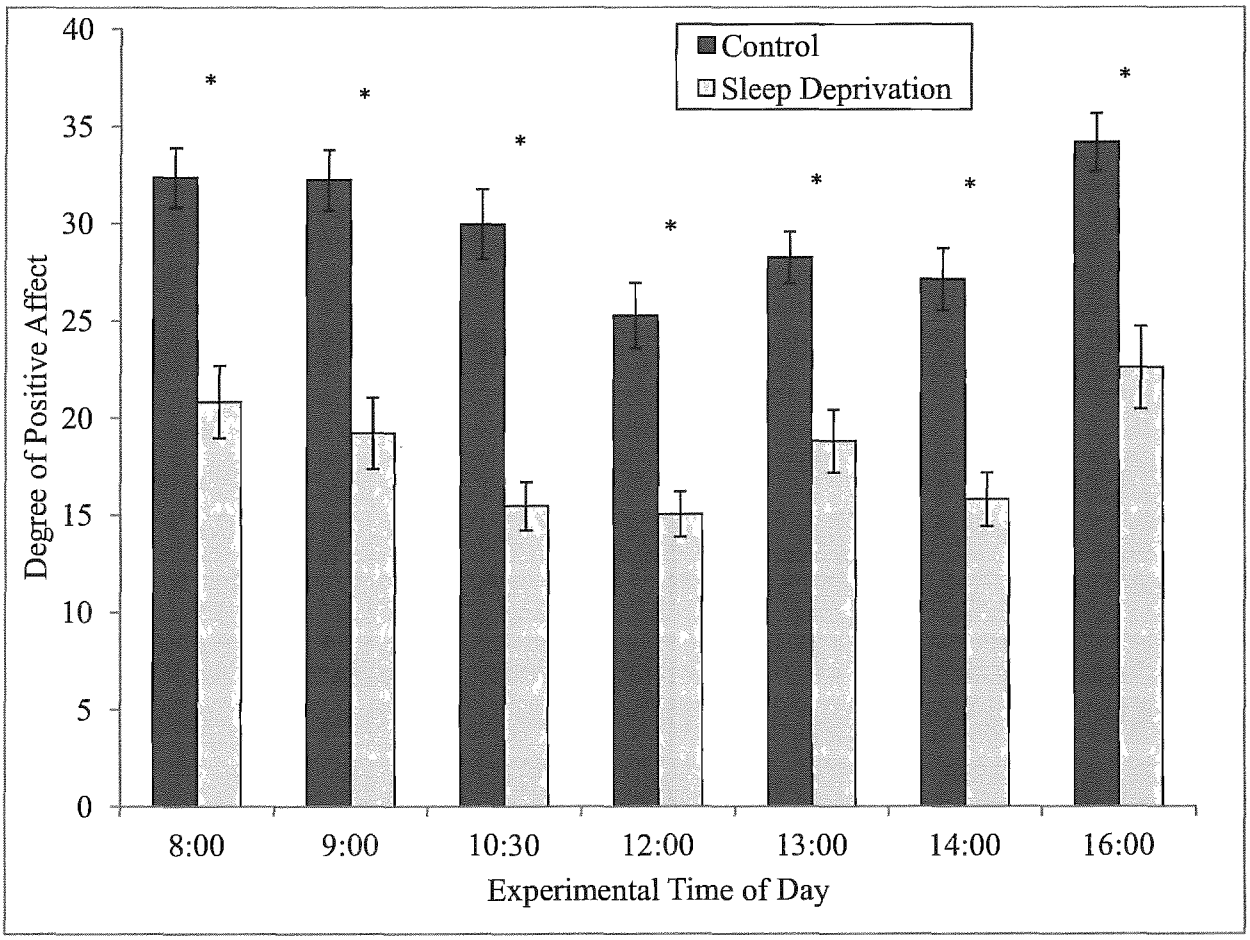


Figure 4. Positive Affect Scale measured by the PANAS. The asterisks* represent significant differences in positive mood on the experimental day and error bars represent standard error of the mean.

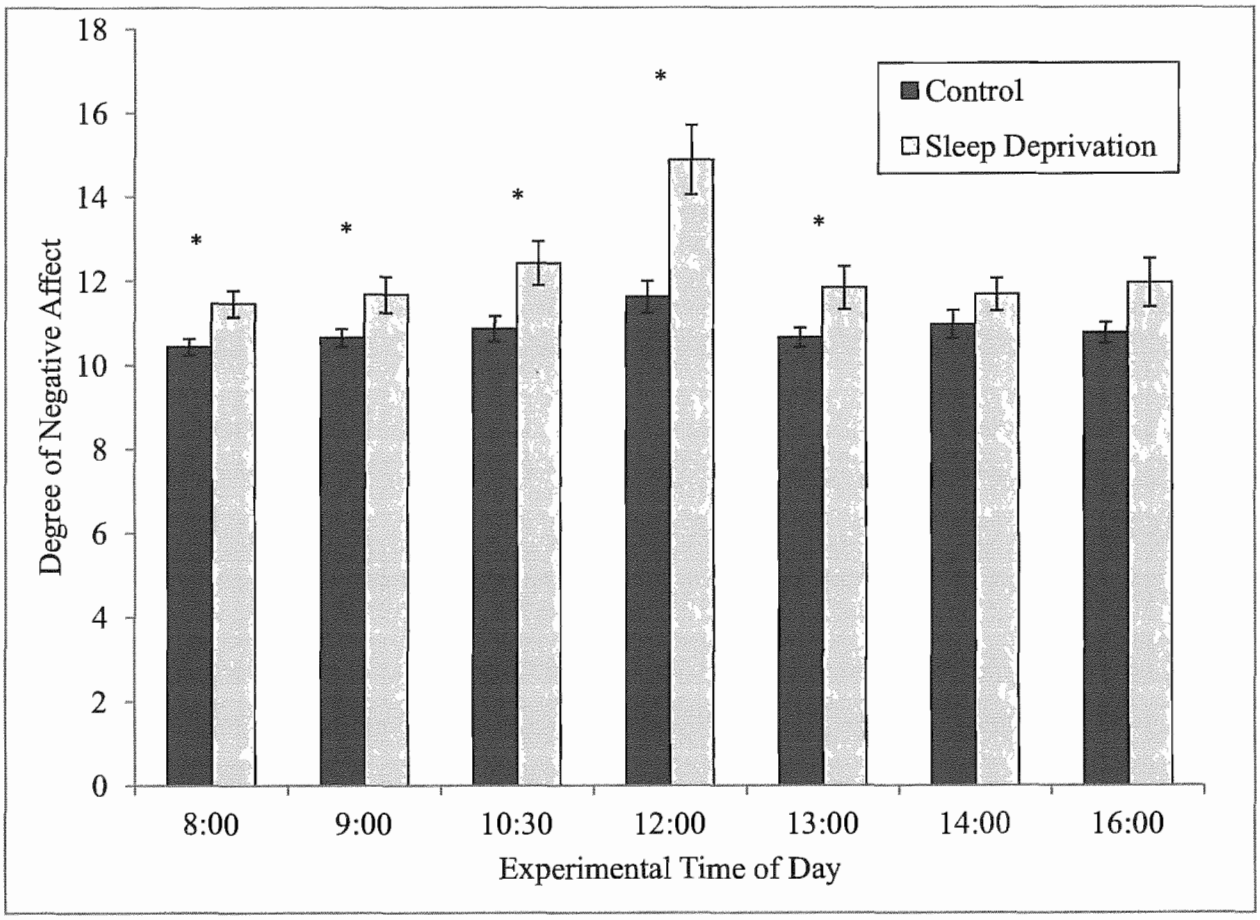


Figure 5. Negative Affect Scale measures by the PANAS. The asterisk* represent significant differences in negative mood on the experimental day and error bars represent standard error of the mean.

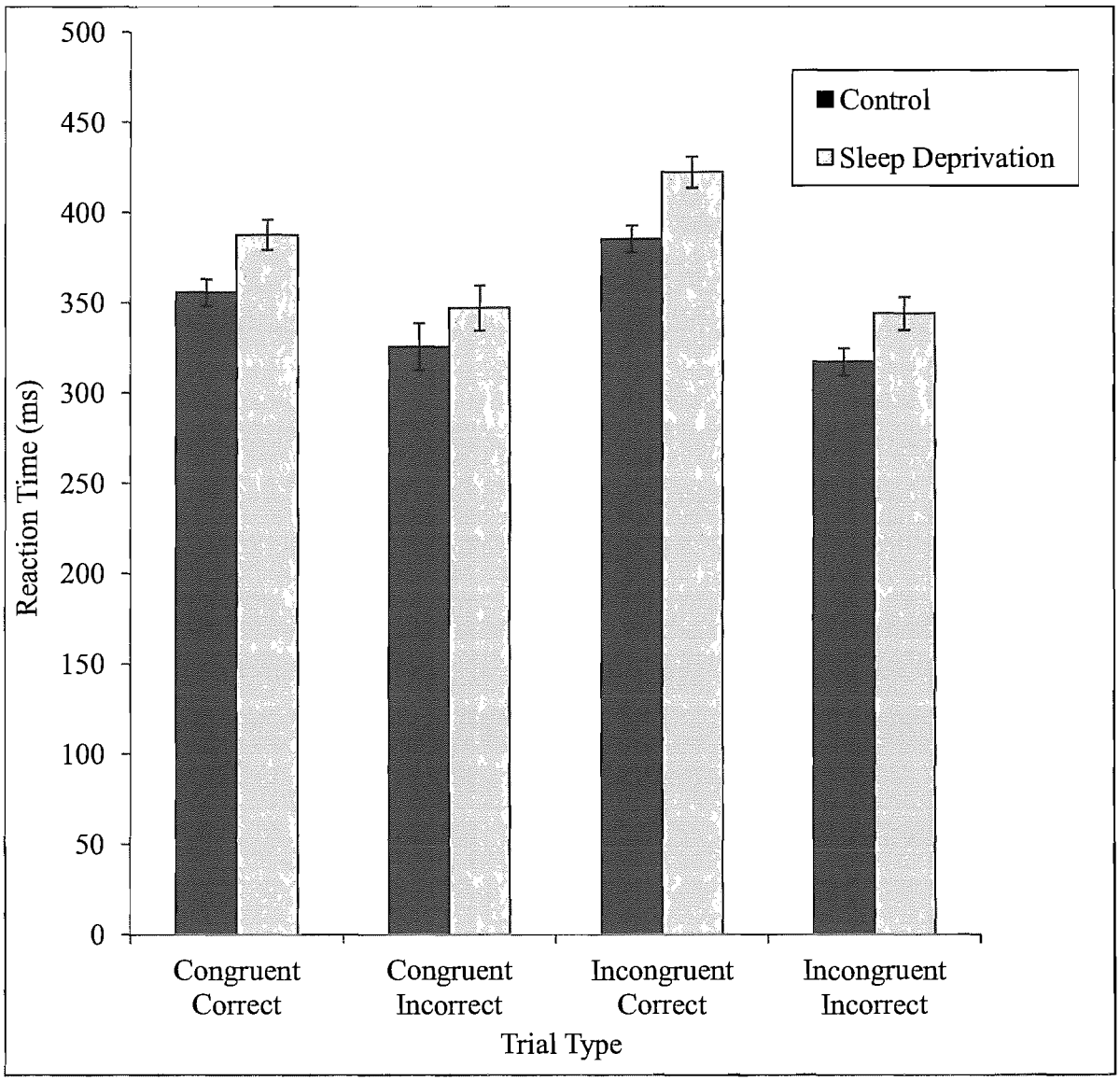


Figure 6. Behavioural reaction times (ms) to both correct and incorrect congruent and incongruent trial types. Error bars represent standard error of the mean.

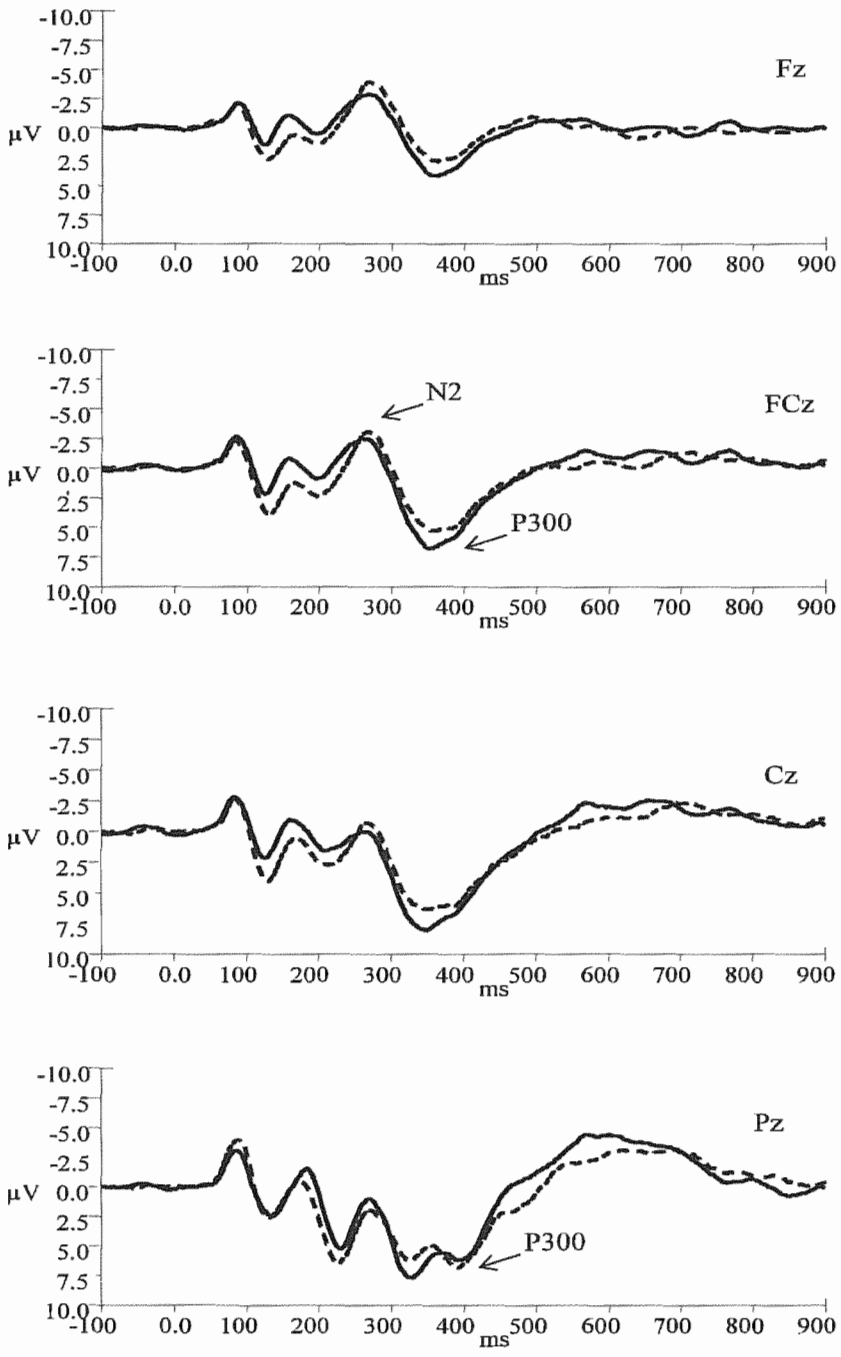


Figure 7. Stimulus-locked averages to correct congruent trials on the Flanker task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent Control group whereas black dashed lines represent Sleep Deprivation group. The N2 deflection is largest at FCz and P300 is largest at Pz. Grand averages are filtered 1-30Hz FIR.

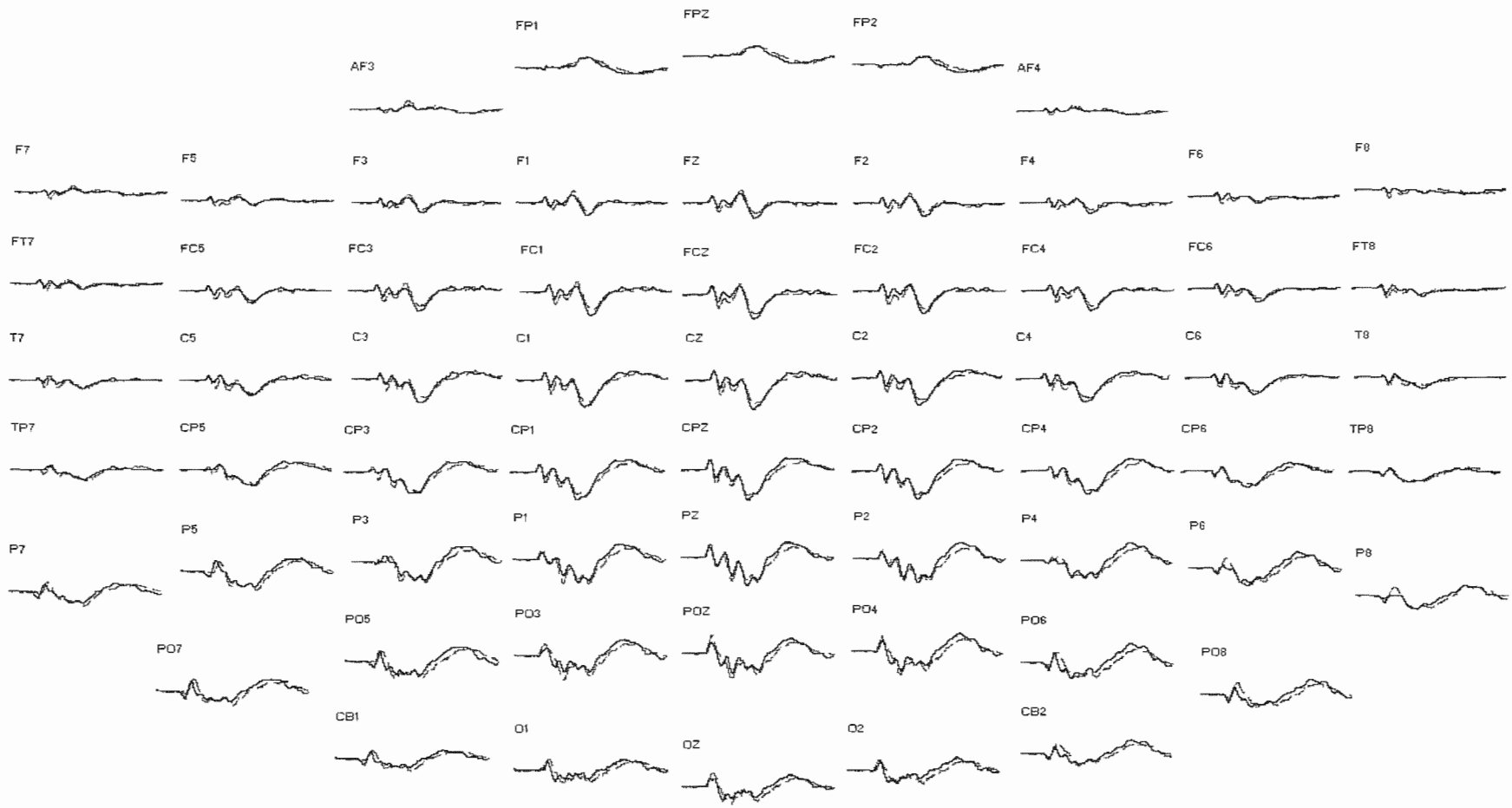


Figure 8. Topography for stimulus-locked averages to correct congruent trials on the Flanker task superimposed between groups. Black solid lines represent Control group whereas grey dashed lines represent Sleep Deprivation group. Sweep time: -100 ms to 900 ms.

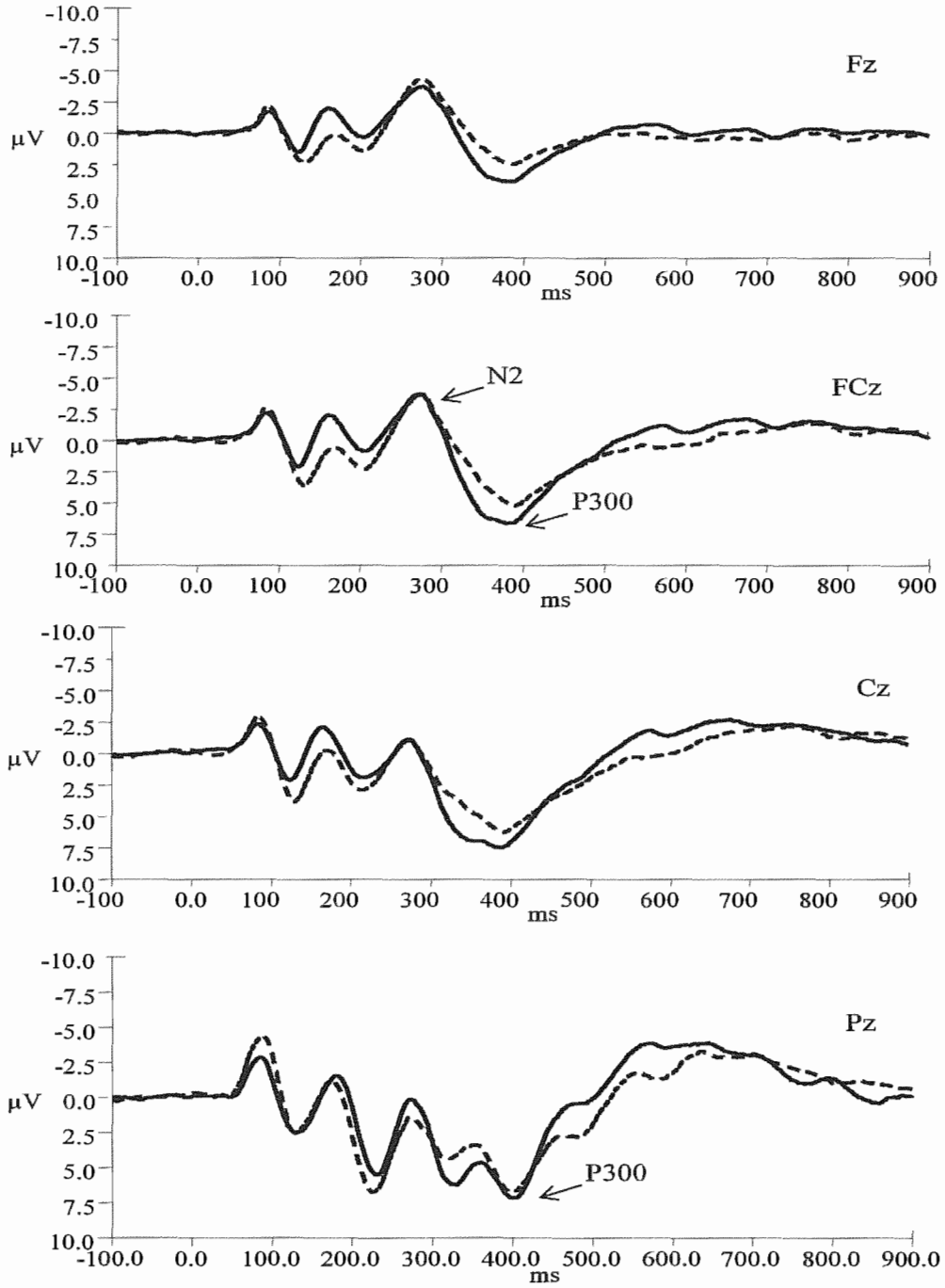


Figure 9. Stimulus-locked averages to correct incongruent trials superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent Control group whereas black dashed lines represent Sleep Deprivation group. The N2 deflection is largest and FCz and P300 is largest at Pz. Grand averages are filtered 1-30Hz FIR.

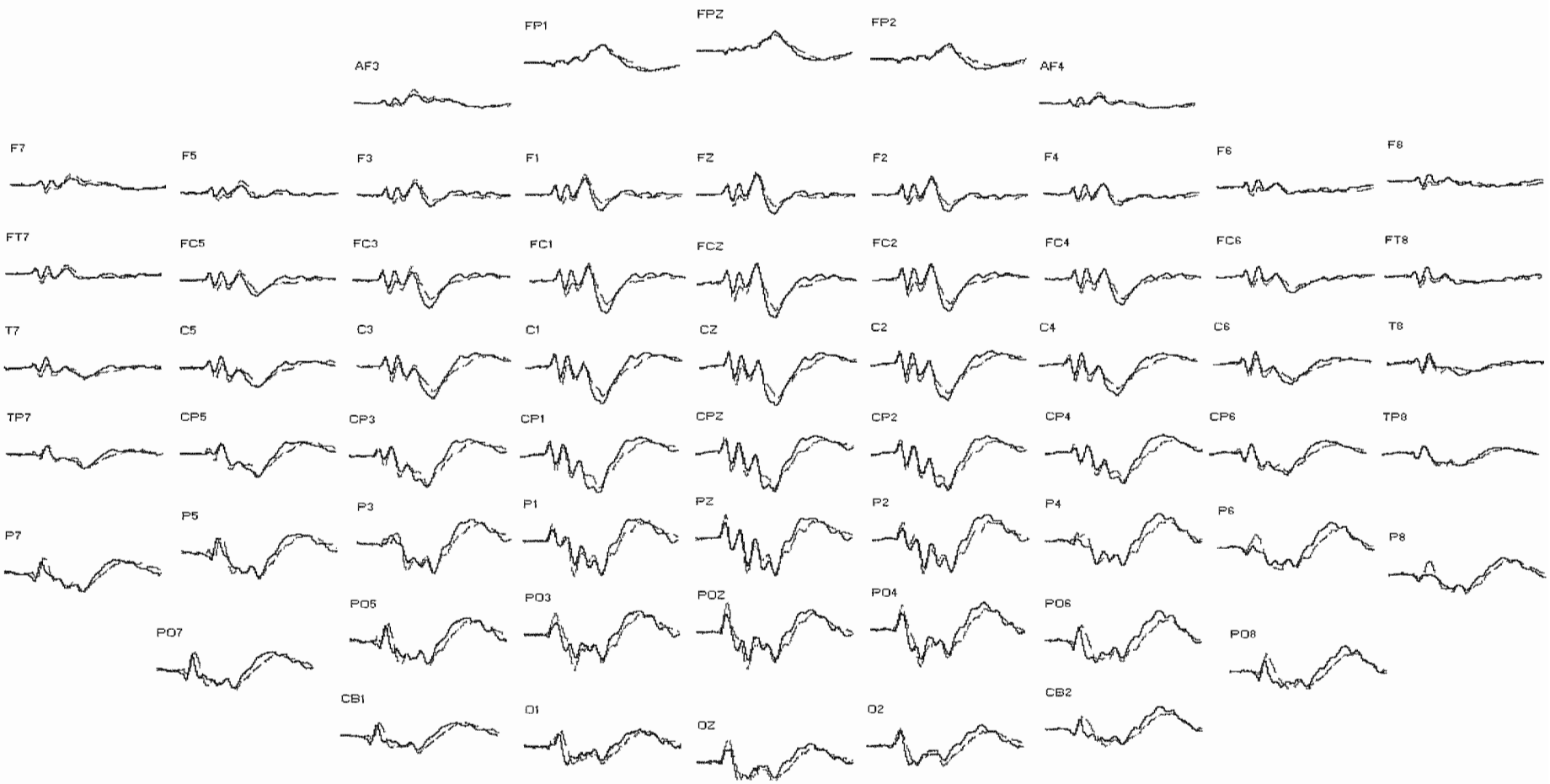
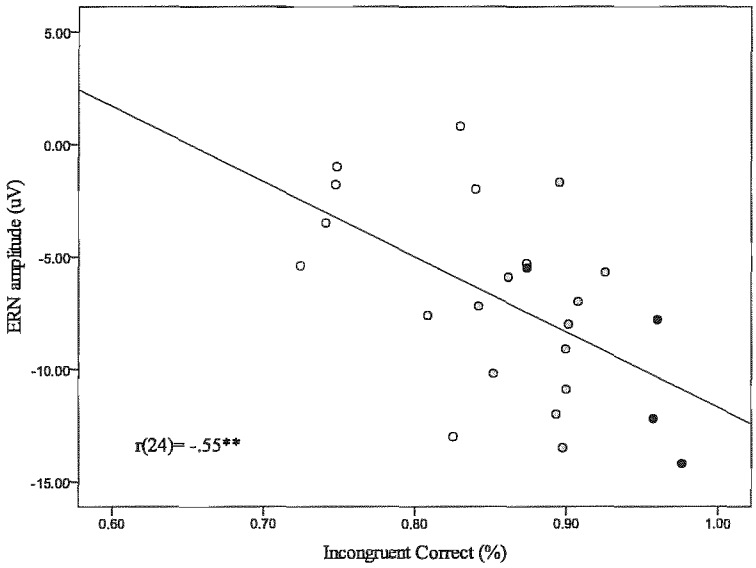


Figure 10. Topography for stimulus-locked averages to correct incongruent trials on the Flanker task superimposed between groups. Black solid lines represent Control group whereas grey dashed lines represent Sleep Deprivation group. Sweep time: -100 ms to 900 ms.

A. Control



B. Sleep Deprived

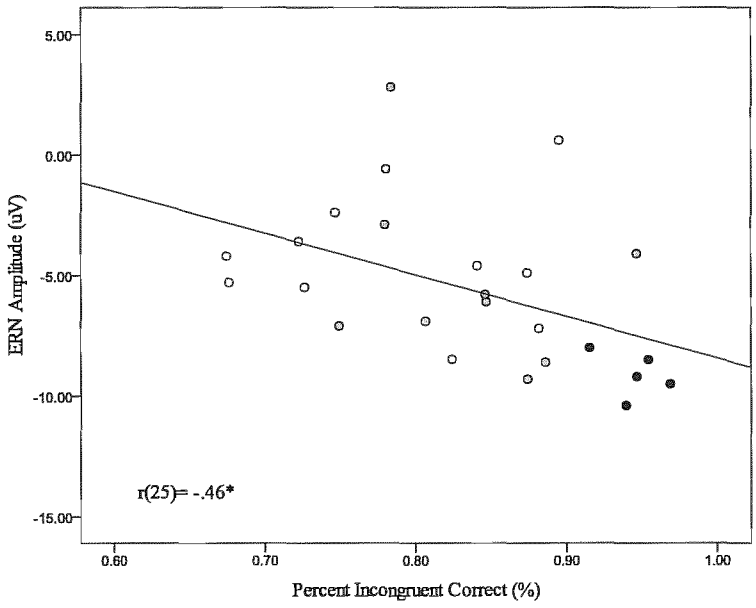
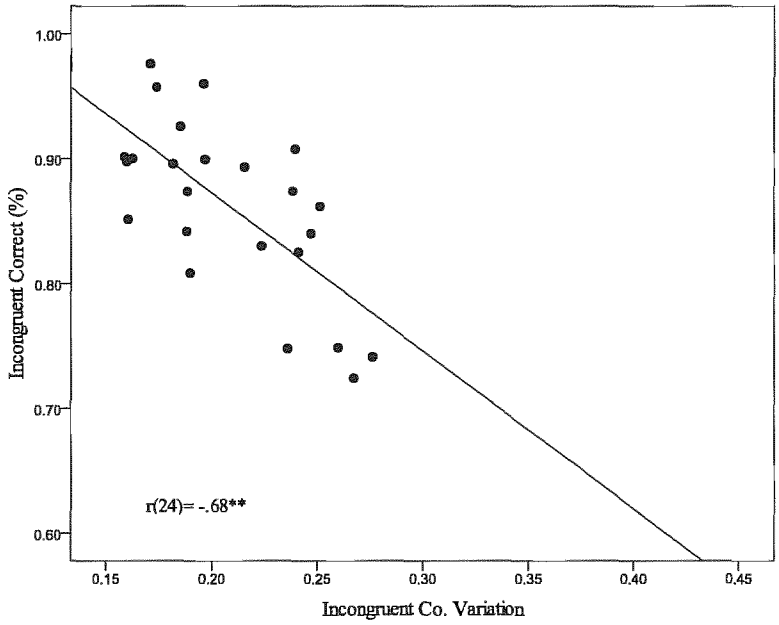


Figure 11. In the Flanker task, ERN amplitude was negatively correlated with response accuracy to incongruent trials in both controls (A) and sleep deprived (B) individuals. (Black+Grey+White) = all cases in original analysis; (Grey+White) = First 20 errors sub group analysis; (White) = First and Last 20 errors sub group analysis. ERN amplitude is smaller with increased number of errors. Note: * $p < .05$, ** $p < .01$.

A. Control



B. Sleep Deprived

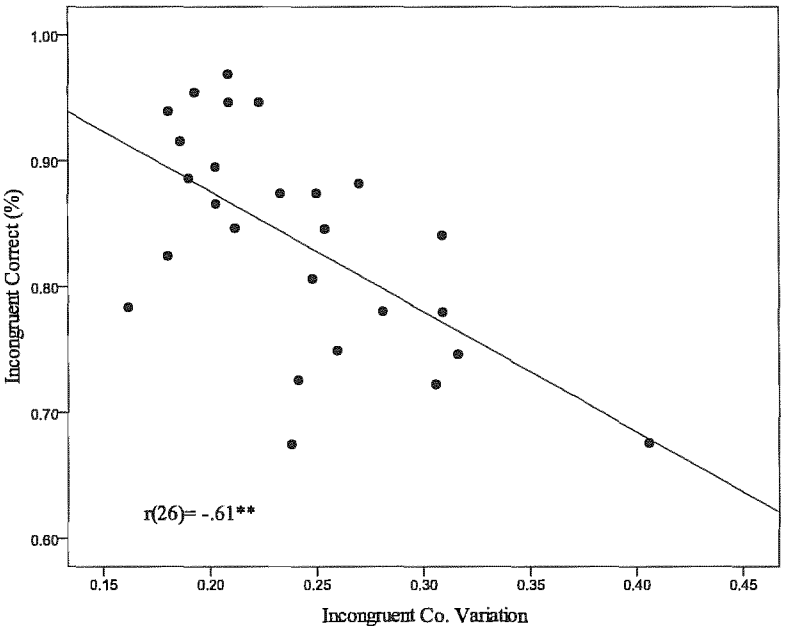


Figure 12. In the Flanker task, incongruent accuracy was negatively correlated with incongruent correct coefficient of variation in both controls (A) and sleep deprived (B) individuals. Poor performance was associated with more response variability. Note: ** p <.01.

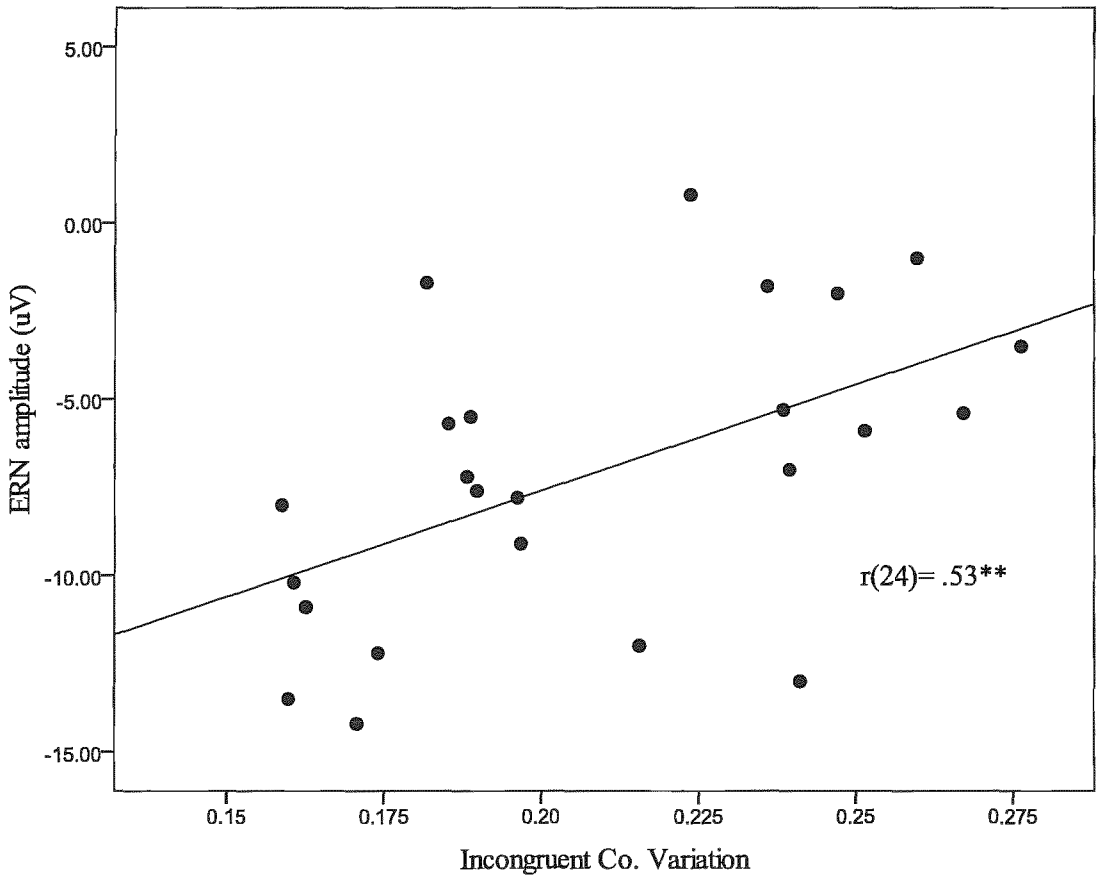


Figure 13. In the Flanker task, ERN amplitude was positively correlated with incongruent correct coefficient of variation in controls. ERN was smaller with larger variability in response time on the Flanker task, in the control group only. Note: ** $p < .001$.

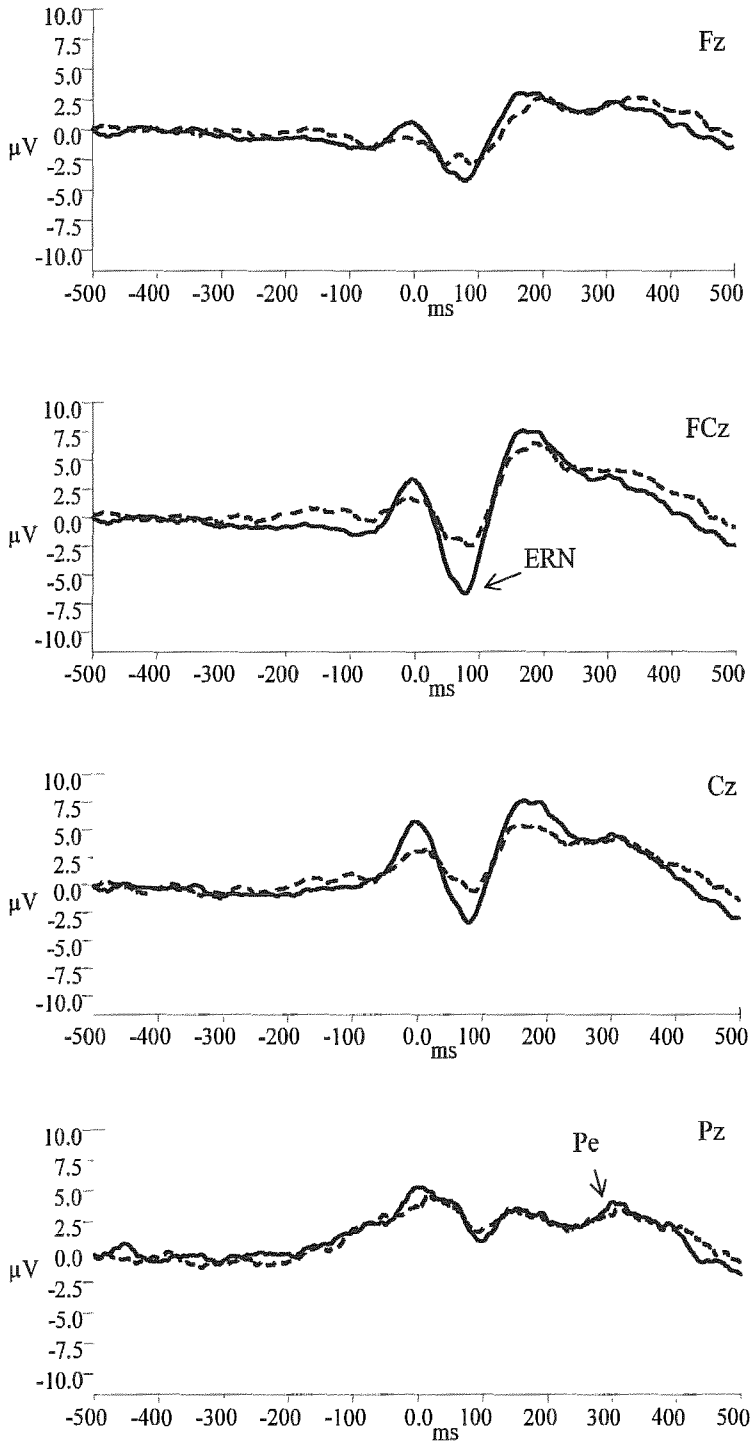


Figure 14. Response-locked averages the first 20 artifact free incorrect responses superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent the Control group whereas black dashed lines represent the Sleep Deprivation group. The ERN deflection is largest and FCz and: Pe is largest at Pz. Grand averages are filtered 1-20Hz FIR.

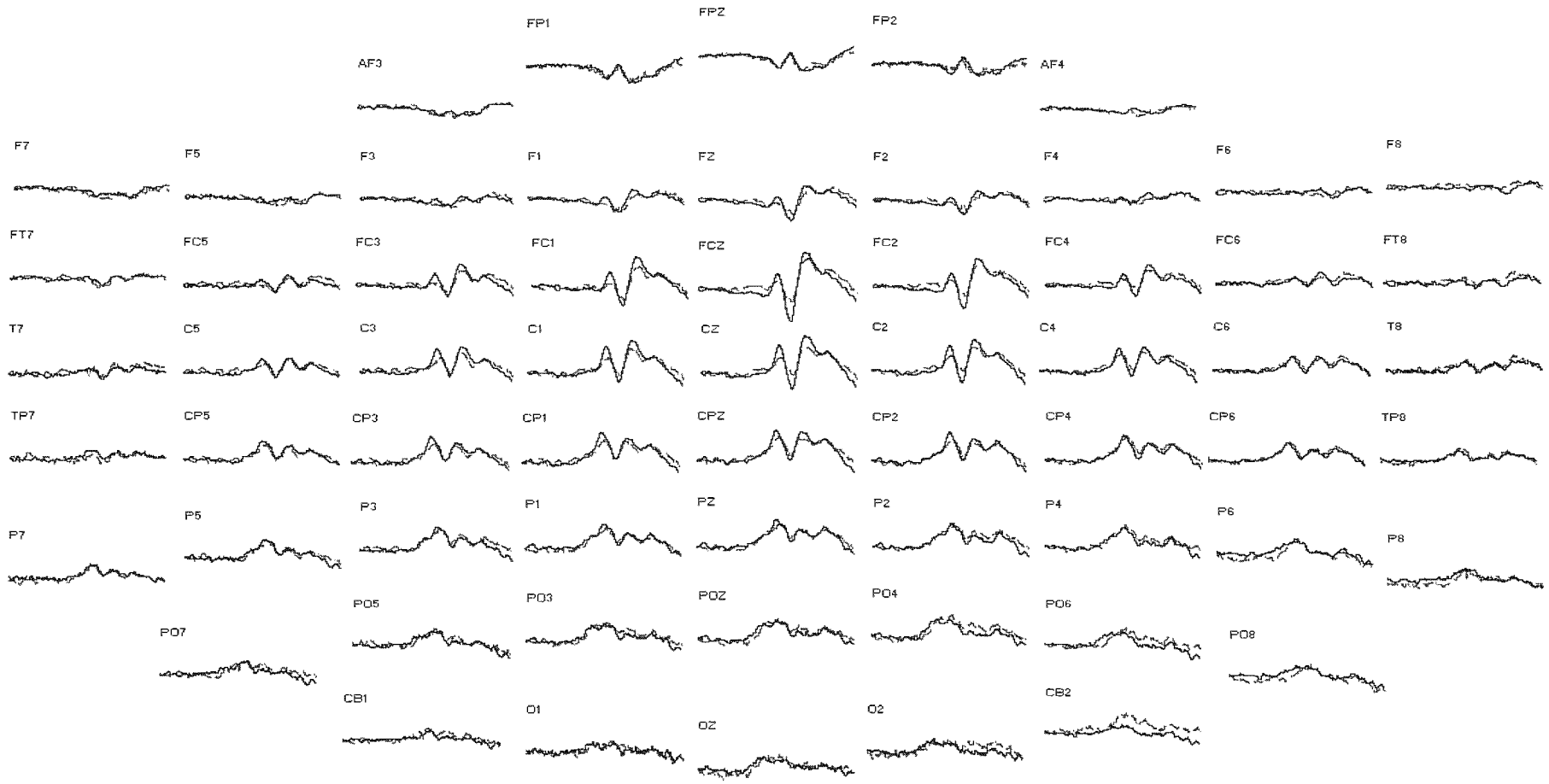


Figure 15. Topography for response-locked averages to incorrect trials on the Flanker task superimposed between groups. Black solid lines represent Control group whereas black dashed lines represent Sleep Deprivation group. Sweep time: -500 ms to 500 ms.

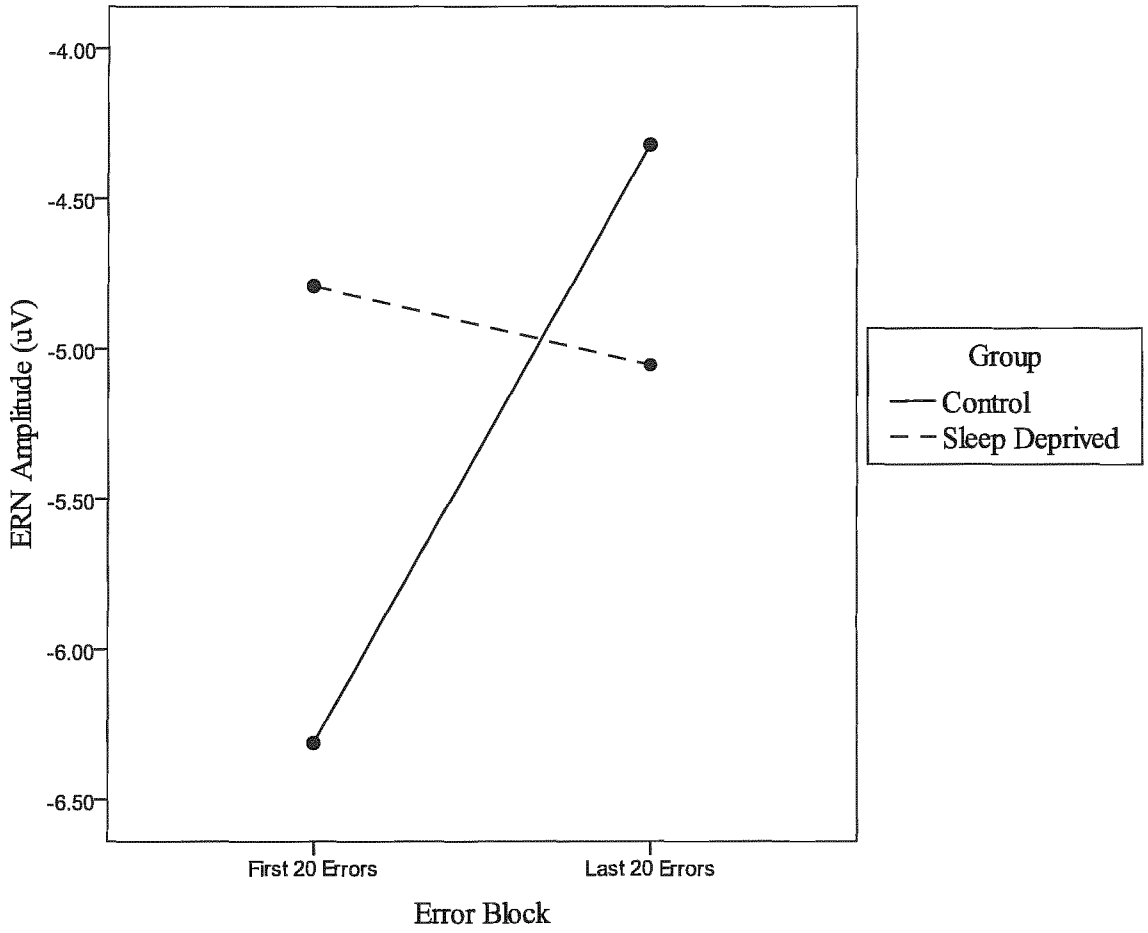


Figure 16. Error-related negativity to the first 20 and last 20 artifact free incorrect responses compared between groups in a sub sample of individuals who made 40+ errors. Note: ERN response is stable across early and late responses in the sleep deprived group, whereas for controls, the ERN response is smaller for the last 20 errors compared to the first 20 errors.

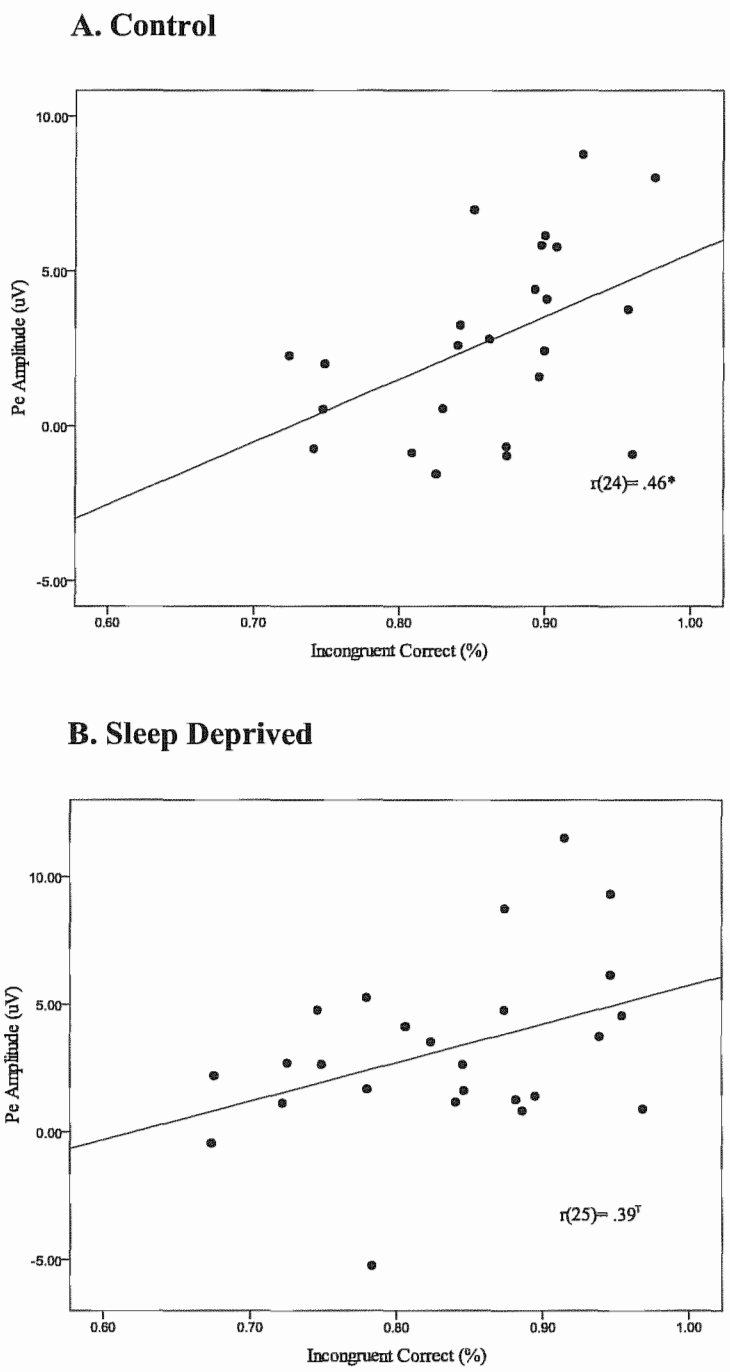


Figure 17. In the Flanker task, Pe mean amplitude was positively correlated with response accuracy to incongruent trials in Controls (A); a trend was apparent in Sleep Deprived group (B). A larger Pe was associated with greater accuracy. Note: * $p < .05$, $^{T}p = .057$.

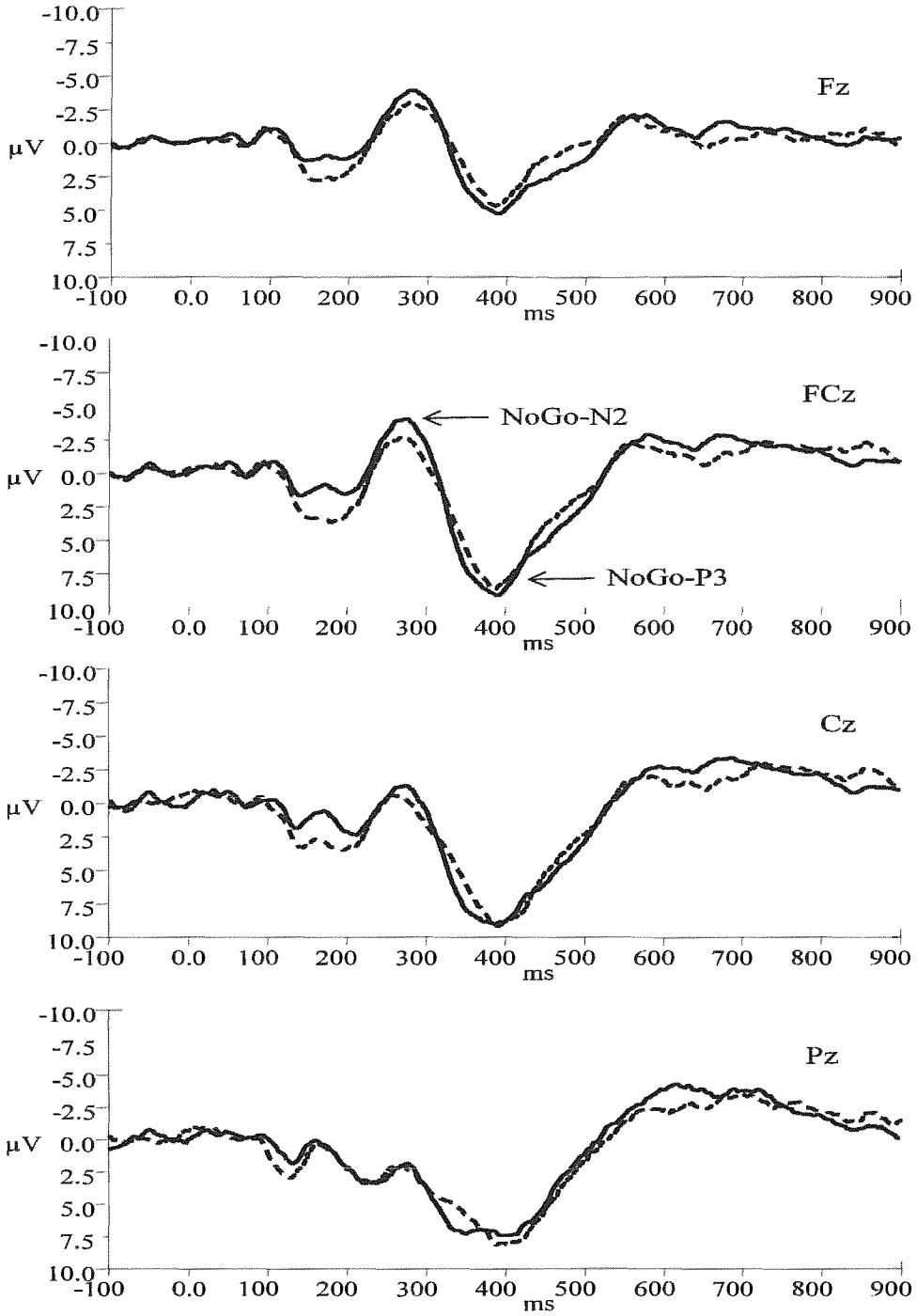


Figure 18. Stimulus-locked averages to correct NoGo responses in a Go/NoGo task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent Control whereas black dashed lines represent Sleep Deprivation. The NoGo-N2 and NoGo-P3 deflections are largest at FCz. Grand averages are filtered 1-30Hz FIR.

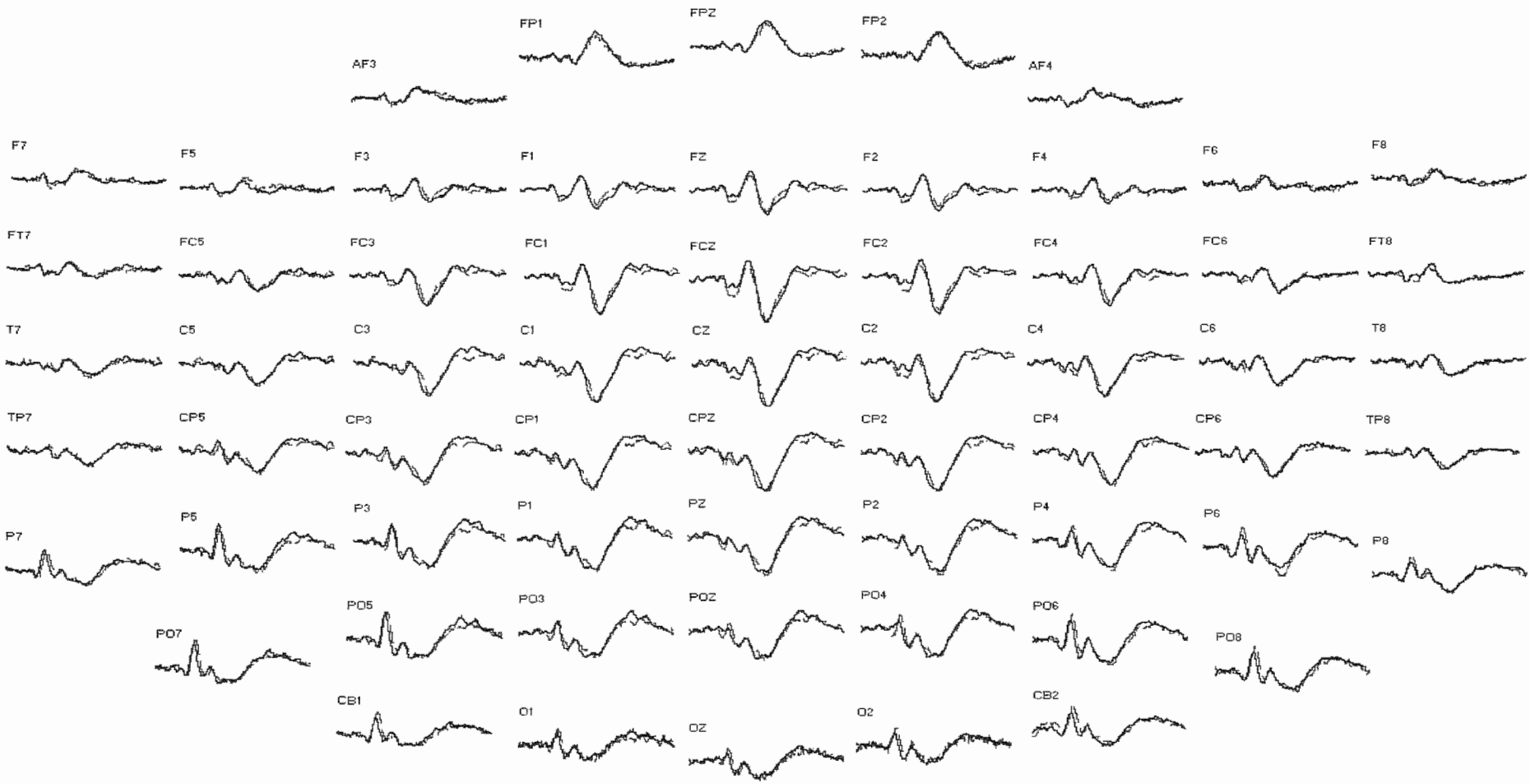


Figure 19. Topography of stimulus-locked averages to correct NoGo responses in a Go/NoGo task superimposed between groups. Black solid lines represent Control whereas grey dashed lines represent Sleep Deprivation. Sweep time -100 ms to 900 ms.

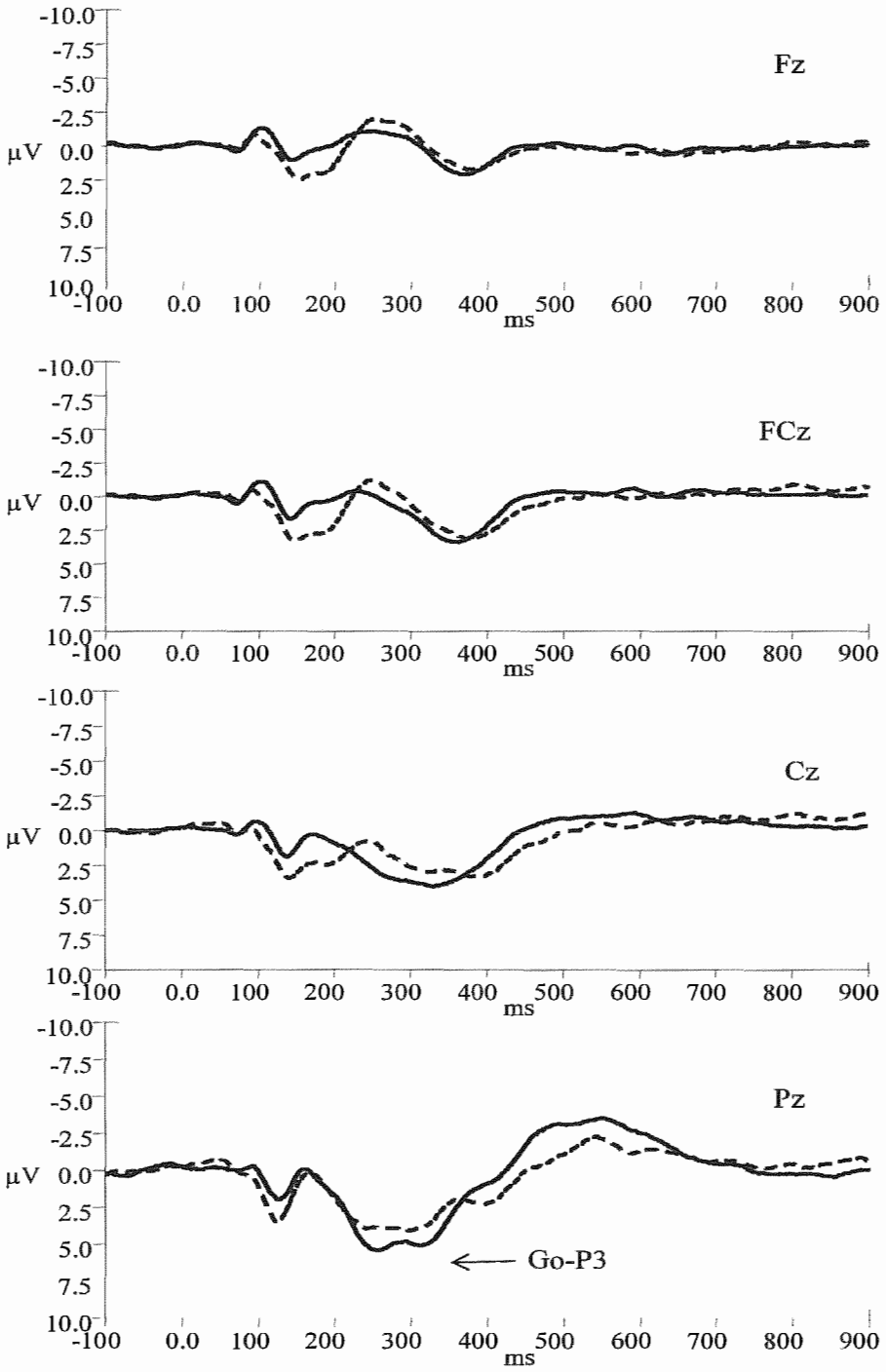


Figure 20. Stimulus-locked averages to correct Go responses in a Go/NoGo task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent Control whereas black dashed lines represent Sleep Deprivation. The Go-P3 deflection is largest at Pz. Grand averages are filtered 1-30Hz FIR.

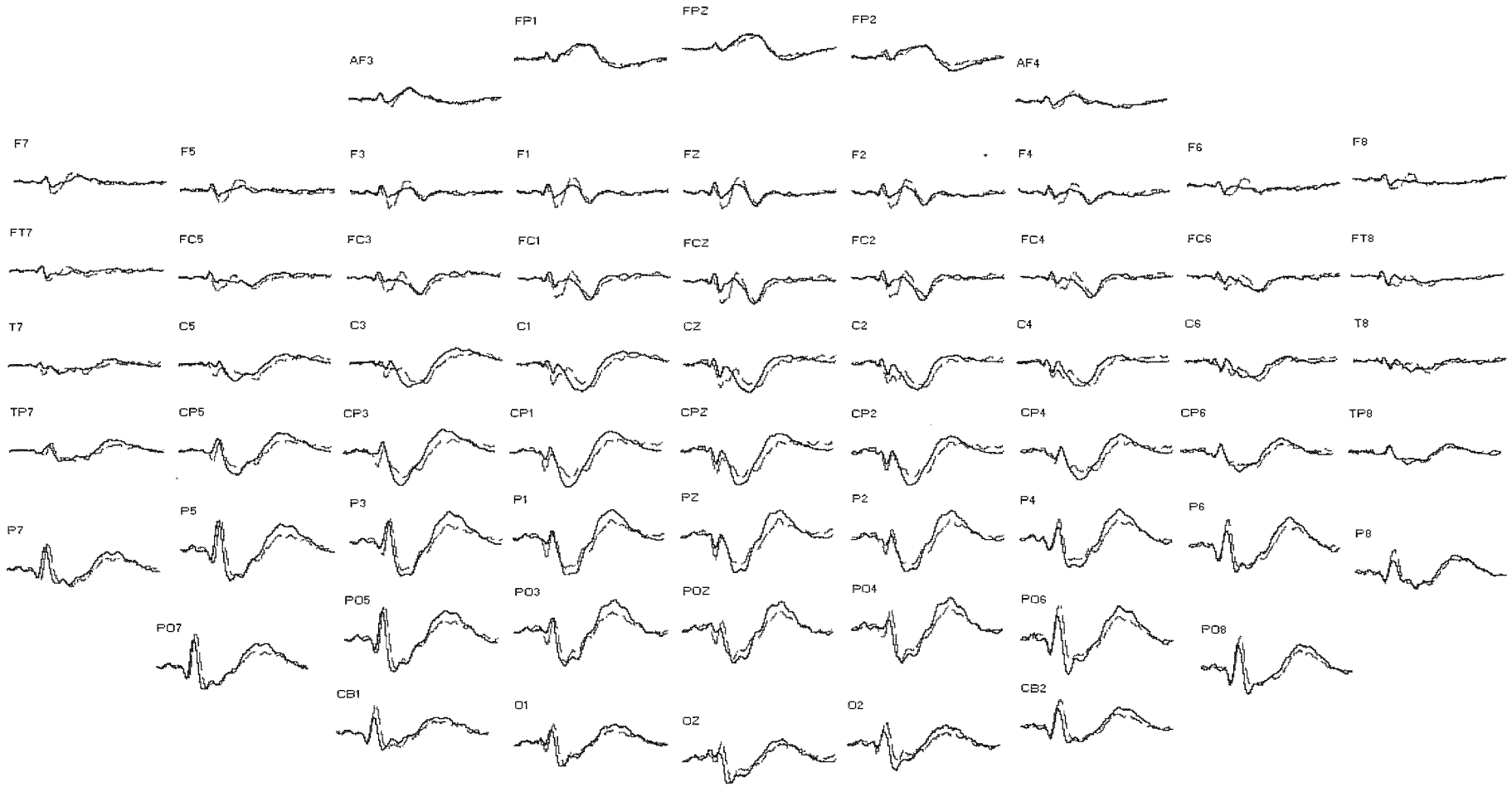


Figure 21. Topography of stimulus-locked averages to correct Go responses in a Go/NoGo task superimposed between groups. Black solid lines represent Control whereas grey dashed lines represent Sleep Deprivation. Sweep time -100 ms to 900 ms.

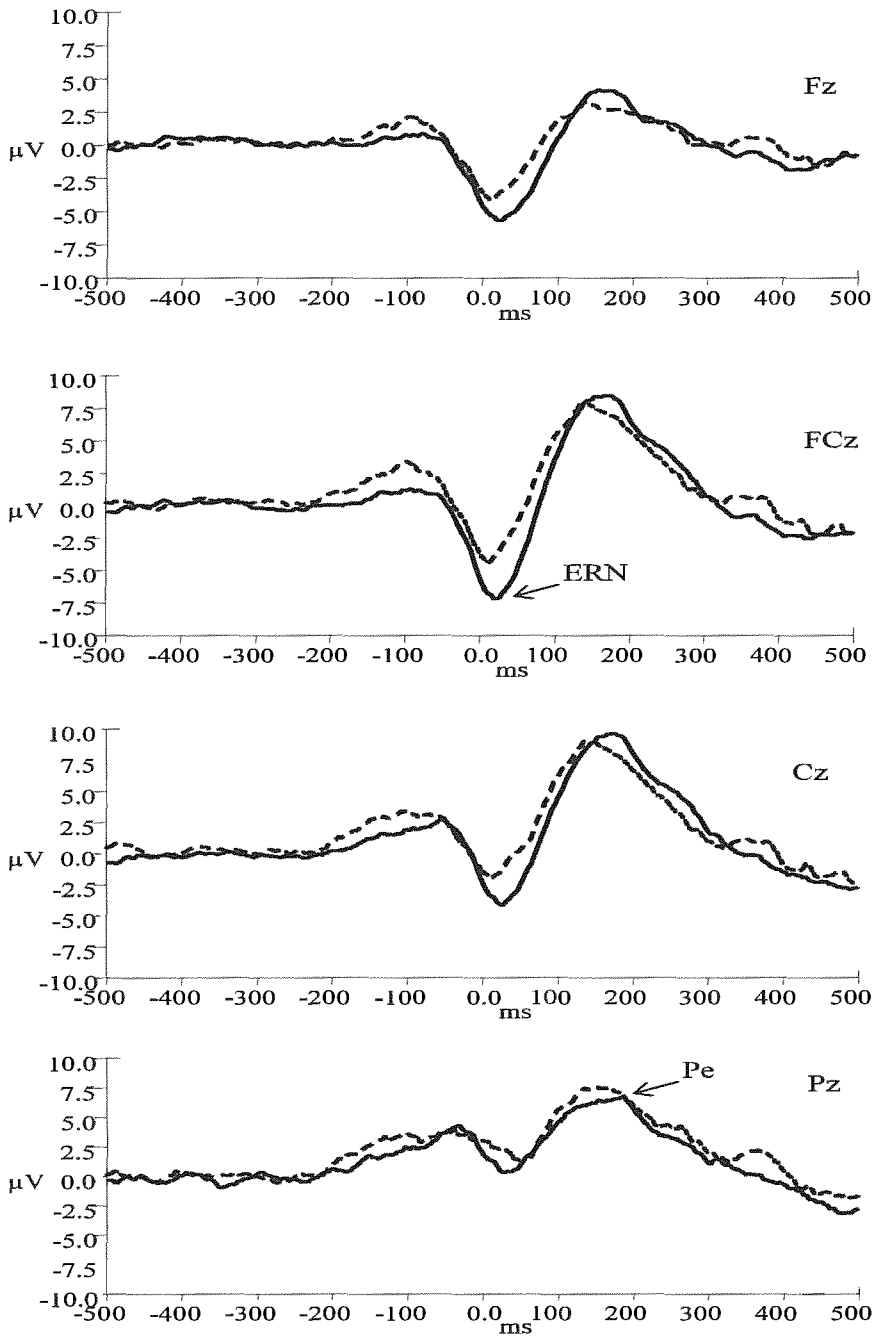


Figure 22. Response-locked averages to incorrect responses in a Go/NoGo task superimposed between groups. Midline Fz, FCz, Cz, and Pz are illustrated above. Black solid lines represent Control whereas black dashed lines represent Sleep Deprivation. The ERN deflection is largest and FCz and: Pe is largest at Pz. Grand averages are filtered 1-20Hz FIR.

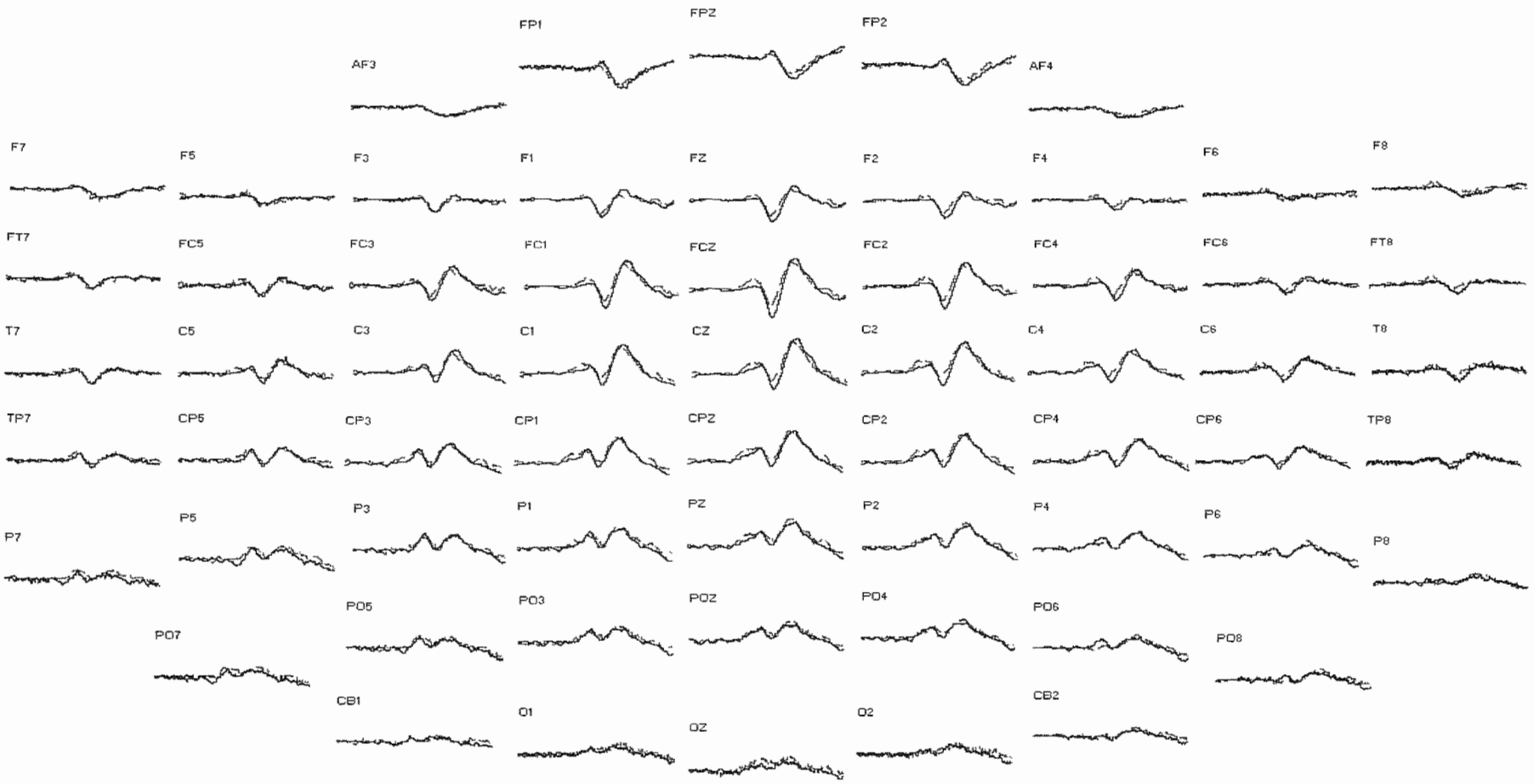


Figure 23. Topography of response-locked averages to incorrect NoGo responses in a Go/NoGo task superimposed between groups. Black solid lines represent Control whereas grey dashed lines represent Sleep Deprivation. Sweep time -500 ms to 500 ms.

Appendix A

HOW DO YOU FEEL?

CALM ——— IRRITABLE

HAPPY ——— SAD

ENERGETIC ——— SLUGGISH

RELAXED ——— TENSE

STANFORD SLEEPINESS SCALE

Please check (✓) the statement which best describes your state of sleepiness. (Choose only **ONE** statement)

	1	Feeling active, vital, alert, or wide awake
	2	Functioning at high levels, but not at peak; able to concentrate
	3	Awake, but relaxed; responsive but not fully alert
	4	Somewhat foggy, let down
	5	Foggy; losing interesting in remaining awake; slowed down.
	6	Sleepy, woozy, fighting sleep; prefer to lie down
	7	No longer fighting sleep, sleep onset soon; having dream-like thoughts

Appendix B

Telephone Interview Sleep Deprivation 2009 Study

Date:	**Time:**

I. DESCRIBE STUDY:

We are interested studying the effects of sleep loss on daytime performance. In this study, we will record your sleep patterns at night and your performance on cognitive tasks during the day. The entire study involves 4 phases:

1. a 1-hr orientation where you will tour the facilities, complete questionnaires on your sleep/wake habits, and undergo a hearing test;

2. an overnight sleep study to screen for sleep disorders;

3. Completing a sleep diary at home for a period of 7-14 days prior to participation in the study, which provides information on your sleep times, caffeine intake, and time of major meals and activities;

4. the main part of the study involves spending 2 consecutive nights and 1 day in the Sleep Laboratory. On the first night, everyone gets to sleep for 8 hrs and you are free to leave the lab during the day (but must refrain from naps, exercise, caffeine, alcohol and nicotine that day). On the 2nd night, you may or may not be sleep deprived. If you are in the sleep deprivation group, you will be asked to remain awake for a period of approximately 35 hours. All participants will perform a 2 ½ - hour battery of tests on day 2 to measure mood, alertness, and attention. You will be free to go at about 6pm that day.

You will be given $75 for complete participation in the study.

Are you interested? [yes] – Ok, I have a few questions for you to make sure you are suitable for the study. If you are the type of person we are looking for, we will then have you come in for an information session, where you can see the Sleep Lab and ask questions about the study. You can decide at that time if you like to participate in the rest of the study.

II. INCLUSION CRITERIA:

Would you be **available** to participate (indicate schedule): _____
Age (18-30): _____
Weight (in kg): _____
Gender: (Circle): M / F
Smoker: Y / N
Handedness: R / L
How many caffeinated drinks do you typically have in a day [min - moderate, <3]: _____
Is English your first language (if not, did you learn before age 8 or describe fluency): ___
Do you have any difficulties with hearing? [no, in both ears]: _____

III. Questions on SLEEP:

1. Do you consider yourself to be a good sleeper? [yes]:_____
2. What are your usual sleeping times (e.g., bedtime and rise time) [23:00-07:00]:

3. How does this change on weekends? [sleeping-in a bit is ok]:

4. Do you have difficulty *falling* asleep at night [no]:

5. Do you *wake up* often during the night and are unable to return to sleep [no]:

6. Have you ever been diagnosed with a Sleep Disorder [no]:

7. Would you describe yourself as *excessively* tired during the day [no]:

8. Do you currently work shift work [no]; any history of shiftwork?

9. Do you take daytime naps? Y / N
   How frequently (# / week) _____ Duration for each _____
10. Have you ever pulled an all-nighter? How often/how many times etc?

IV. Questions on HEALTH:

1. Are you presently in good health [yes]:

2. Taking any medications [no, except BCPs]:

3. Any history of depression, anxiety or schizophrenia [no]:

4. Any history of head injury (e.g., car accident, stroke, loss of consciousness), epilepsy, or other neurological condition [no]:

5. Any history of chronic pain or Raynaud's Syndrome (extreme whitening and severe pain in the hands with mild cold) [no]:

6. Any history of heart disease or cardiac abnormalities [no]:

CONTACT INFORMATION:

Name:	
Telephone Number(s)	
E-mail:	
Best time/ method for contact:	
Date for Orientation:	

Note: ask about dreadlocks or braids as participation will NOT be possible due to EEG cap hook-up

Appendix C

Letter of Information / Consent Form
Sleep Research Laboratory
Psychology Department, Brock University

Title of Study: Effects of Sleep Loss on Human Performance

Principal Investigator: Kimberly A. Cote, Ph.D.
Co-Investigators: Ryan Renn, B.Sc., Cathy Mondloch, Ph.D., Cheryl McCormick, Ph.D.

This letter of information/consent form is provided to you for your information on the website of the Brock University Sleep Research Laboratory. You should carefully read this form to understand all aspects of participation in the research study prior to completing the on-line eligibility questionnaires. By completing the on-line questionnaires, you are acknowledging that you have read and understood this form and you are providing consent to participate in the full research study. You will be asked to sign this form and be given a copy during your next visit to the Sleep Laboratory.

If you have questions about the details of this study prior to completing the on-line questionnaires, please call the Sleep Laboratory at 905-688-5550, ext.3795.

Name of Participant:
(*Please print your name in the space above on the paper copy only*)

PART A: INFORMATION ABOUT THE STUDY

I understand that I am being invited to participate in a research study investigating attention and arousal following one night of sleep loss. This study will be of benefit to me because I will be able to learn about the impact of sleep loss on performance; as well, it will inform the scientific community about the impact of sleep on waking brain function.

Specifically, my participation will involve first **completing on-line screening questionnaires** on my sleep habits and personality characteristics. If eligible, I will be contacted and asked to keep a **sleep diary for a 1-2-week period** while at home (these forms will be provided to me or I can enter the data on-line). I understand that there is no honorarium provided for my participation in the pre-study activities described above.

I understand that I will then be required to **spend one night in the Sleep Laboratory** for the purpose of screening for sleep disorders. At this time, I will be given a copy of this Information/Consent form to sign, and I will undergo a hearing test to ensure I have hearing within normal range. My sleep will be recorded by placing electrodes on my scalp to measure brain wave activity (applied with water-soluble paste, not glue), electrodes by my eyes to measure eye movements, and electrodes under my chin to measure muscle movement. Electrodes will also be placed on my legs to test for unusual

leg kicks during sleep. In addition, bands that are placed around my chest and waist will monitor my breathing during the night. Finally, my heart rhythms will be recorded to check for normal heart rhythms during sleep. I understand that if I am found to have any sleep, breathing, movement, or cardiac abnormalities, I will not be asked to participate in the remainder of the study, but will be paid for one night in the laboratory ($10). If I am suspected of having a sleep disorder, it will be recommended that that I seek a full diagnostic assessment at a Sleep Disorders Clinic. If I am excluded from further participation, my data will be destroyed.

I understand that if selected to participate in the main study, I will then be scheduled to **spend two nights and one day in the Sleep Laboratory**. I may be assigned to the Sleep Deprivation group who will be asked to remain awake for approximately 36 hours. Alternatively, I may be assigned to the control group who will sleep on both nights.

On Night 1, everyone will sleep undisturbed overnight (from 11pm to 7am). My sleep will be recorded as described above. I will need to arrive at 8pm.

On Day 1, I will be free to leave the laboratory after 8am, but must refrain from naps, exercise, caffeine, alcohol and nicotine. I return to the lab that evening at 8pm.

On Night 2, I may be asked to sleep or to go without sleep between 11pm and 7am. The assignment to the Sleep Deprived or Sleep Group will be based on random assignment.

On Day 2, I will be required to remain in the laboratory all day and perform computer assessment batteries from 10:30-11:45 and 2:00-3:30. Examples of the tasks include: reaction time, memory, and processing of emotional pictures and faces. I understand that I will have been shown all of these computer tasks during a demo session on the screening night and will have the opportunity to practice tasks on the baseline night.

During the performance assessment, I will have my brain activity monitored with a number of electrodes placed across the scalp. These electrodes are applied using an electrode cap, which fits like a swimming cap. I will also have my eye movements, muscle activity and heart rate monitored.

At 3:30 on Saturday the electrode cap will be removed, and I will participate in one more task. I will play a computerized game of competition that is designed to examine the effects of sleepiness on game playing. Before and after the 30-minute game, I will be asked to provide hormone samples by spitting in a test tube for a measure of stress.

On Day 2 (Saturday), no naps or exercise will be permitted. I will also not be permitted to use a computer, cell phone or other electronic devices. I may use the telephone in the laboratory to make brief calls if necessary. I will be permitted to

read, watch movies, and play cards or board games. Meals will be provided to me on Day 2 in the Sleep Laboratory. If I have any food allergies or preferences, I understand that I will have opportunity to communicate this to the research assistant ahead of time.

PART B: INFORMATION ABOUT STUDY RISKS AND YOUR RIGHTS AS A PARTICIPANT

I understand that I may experience some skin irritation (redness and dry skin) as a result of having electrodes attached to my scalp and face. This is temporary and may be reduced by applying moisturizing cream to the areas where electrodes were placed.

I understand that I will be asked to go without caffeine for the duration of the study (from the morning of night 1 through to the end of Day2). If I am a heavy caffeine user, I may experience symptoms of caffeine withdrawal such as headache. If I experience headache, I may ask the research assistant for Tylenol medication.
If assigned to the Sleep Deprived group, I understand that it is expected that I will feel very sleepy at times during the study. I understand that a research assistant will be with me at all times (except during washroom breaks), and that this person will talk to me and interact with me continually in order to keep me awake (e.g., play cards). I understand that I may not use prescription or over-the-counter medications with caffeine or other stimulants at any time during the study. Sleep loss sometimes leads to mild, temporary stomach ache or acid reflux; if I experience any of these symptoms, I understand that I may ask the research assistant for an over-the-counter medication (e.g., TUMS). I understand that it is pertinent that I go home immediately after the study ends to sleep. I understand that I should not drive home myself as sleep loss impairs driving performance. If I cannot arrange for a friend or family member to escort me home, I may have the research assistant walk me home if I live in residence on-campus, or I will be provided a taxi ride home. I understand that I should not attempt to work or drive before obtaining enough sleep to feel rested and alert. Specifically, I should go to sleep immediately upon getting home, by 7 or 8 pm at the latest, and sleep through until morning to achieve a sufficient amount of sleep (i.e., get 10-12 hours of recovery sleep). Following the recovery sleep period, I understand that I should still exercise the usual caution one would if experiencing sleepiness, e.g., if I should feel any residual effects or I was not able to sleep well during the recovery sleep.

I understand that the Sleep Laboratory facilities are under 24-hour video surveillance. All activities in the main laboratory, bedrooms, and the kitchen/lounge areas are recorded and stored in the Sleep Laboratory until completion of the study. Because the bedrooms are also under video surveillance, you should note that there will be a lack of privacy while sleeping. The videotaped data will not be used for any research purposes, presentation of data, or advertising.

Upon completion of the entire study (screening, two nights, and one day), I understand that I will receive $100. If I elect to use a portion of this study toward research

participation credit in a Brock course, I will receive the credit plus $80 (that is, less the amount of one night's compensation). Please note that screening procedures are not eligible for credit; participants must enrol in the study to receive credit.

If I choose to withdraw from the study for any reason, and/or I am removed from the study by investigators (e.g., due to sleep disorder or technical problems which prohibit the study from continuing), I will be compensated according to the following schedule:

No Compensation - Pre-study screening (includes online questionnaires, home diaries)
$10 - screening (overnight sleep study)
$20 - Night one of the main protocol **(or the option to take course credit)**
$20 - Night two of the main protocol
$20 - Day two of the main protocol (Saturday when performance assessment and EEG recordings take place)
$30 - Completion bonus.

Please note that the completion bonus is not given because it is expected that it will become more difficult for you to remain in the study over time (i.e., harder to stay awake). It is important that participants understand that we do not obtain any usable data for our research until the final day of the study when the performance assessment batteries are finished.

I understand that my participation is voluntary and I may withdraw from the study at any time, for any reason, without penalty. I am under no obligation to answer any question or participate in any aspect of this project that I consider invasive, offensive, or inappropriate. I understand that I may ask further questions at any time. If I am in the sleep deprived condition and withdraw early, I will be required to sleep in the Sleep Laboratory until I feel refreshed, and I will still require an escorted ride home.
I understand that all personal data will be kept strictly confidential and all information will be coded so that my name is not associated with my answers. Only the researcher named above, and research assistants working under supervision of these researchers, will have access to the data. All research assistant will have signed confidentiality agreements. I understand that I am not anonymous in this study because the nature of the study requires that research assistants interact with each participant in the laboratory on a one-to-one basis and have contact information to schedule appointments.

Your signature below indicates that, you are of the age of legal consent (i.e., 18 years or older), you have read and understood the procedures of the study, and you agree to participate.

Participant's Signature_____ Date _____
(to be signed during your visit to the Sleep Laboratory for sleep screening)

PART C: CONTACT INFORMATION

This research is funded by the Natural Science and Engineering Research Council (NSERC) of Canada. This study has been reviewed and cleared by the Brock Research Ethics Board (File # 09-033). For answers to questions about your rights as a research participant, contact the Research Ethics Officer, at (905) 688-5550 ext. 3035, or reb@brocku.ca.

If you have any questions or concerns about your participation in the study you may contact the Principle Investigator, Dr. Kimberly Cote in the Psychology Department at (905) 6885550, extension 4806.

No individual feedback from the sleep study or performance data may be provided at any time. Feedback about the outcome of the study will be available by request after final publication of the data (email: kcote@brocku.ca).

Please take a copy of this form with you for future reference. IF YOU NEED TO CONTACT THE LABORATORY REGARDING YOUR APPOINTMENT OR STUDY PROCEDURES, PLEASE CALL US AT **905-688-5550, EXT. 3795.**

I have fully explained the procedures of this study to the above volunteer.

Researcher's Signature_____ Date _____

NOTE: Signature below indicates that the above name person has completed the part of the study eligible for research participation credit (e.g., 3 credits in Psyc 1F90).

Researcher's Signature_____ Date _____
(Sleep Technician)

Appendix D

Pre-Sleep Questionnaire

Participant I.D. _____ Time:_____

Date: _____

At what time did you awaken today? _____a.m. p.m.

Has today been an unusual day in any way? No ___ Yes___. If yes, explain:

Did you fall asleep or take a nap today? No ___ Yes___. If yes, when and for how long:

Did you drink any alcohol today? No____ Yes____. If so, when _____
how much? _____

Did you, or will you, use any **medications** (prescription or non-prescription) **today?**
No____ Yes _____. If yes, **specify type and amount**:

Have you used any **prescription** medication in the **last 2 weeks**? No ____ Yes ____
If yes, **specify type and amount**:

Please indicate how many **cups or glasses** of the following that you have consumed today:

___ coffee, ___decaffeinated coffee, ___ tea, ___ cola, ____ chocolate drinks

At what time did you drink your **last** caffeinated beverage? _____a.m. p.m.

Put a check mark (✓) in the appropriate column to indicate if you are experiencing any of the following, **right now**.

	Not al All	Slightly	Moderately	Intensely
Headache				
Unsteadiness				
Faintness				
Breathing difficulties				
Chest pain				
Sweating				
Numbness [specify:]				
Flushing				
Chills				
Heart Palpitations				
Sexual feelings				
Hunger				
Bloating				
Nausea				
Gastric fullness				
Abdominal pain				
Feverishness				
Constipation				
Diarrhea				
Urinary problems				
Blurred vision				
Irritated eyes				
Puffy eyes				
Blacking out of sight				
Noise in ears				
Reduced hearing				
Increased taste sensitivity				
Increased smell sensitivity				
Dry mouth				
Thirst				

Use the chart below to indicate the severity of any pains, aches, or stiffness that you may be experiencing right now.

On the chart a check (✓) in the row labelled '0' indicates no discomfort.
a check (✓)in the row labelled '6' indicates the worst possible discomfort.

	Head	Neck	Shoulders	Upper Limbs	Chest	Upper back	Lower Back	Abdomen	Hips	Lower Limbs
0										
1										
2										
3										
4										
5										
6										

Fatigue Scale

Please check (✓) the statement which best describes your present state of physical energy or fatigue.

	1	Full of energy: enough to tackle my usual physical activities.
	2	Energy level is quite high but not at its peak: most physical activities would pose no problem
	3	Energy level is such that one would prefer to be doing very light or sedentary tasks at this point.
	4	Energy level is adequate for only routine activities at a leisurely pace.
	5	Energy level is such that it would be preferable to rest before doing any routine activity.
	6	Energy level is quite low: would strongly prefer to rest rather than do anything else
	7	Totally physically exhausted: unable to undertake the least activity.

HOW DO YOU FEEL?

CALM ——— IRRITABLE

HAPPY ——— SAD

ENERGETIC ——— SLUGGISH

RELAXED ——— TENSE

STANFORD SLEEPINESS SCALE

Please check (✓) the statement which best describes your state of sleepiness. (Choose only **ONE** statement)

	1	Feeling active, vital, alert, or wide awake
	2	Functioning at high levels, but not at peak; able to concentrate
	3	Awake, but relaxed; responsive but not fully alert
	4	Somewhat foggy, let down
	5	Foggy; losing interesting in remaining awake; slowed down.
	6	Sleepy, woozy, fighting sleep; prefer to lie down
	7	No longer fighting sleep, sleep onset soon; having dream-like thoughts

Post-Sleep Questionnaire

Participant I.D. _____ Time:_____

Date: _____

Please complete immediately upon the final awakening

How long did it take you to fall asleep last night: _____ minutes

How much sleep do you think you got last night: _____hours

Please indicate with an **X** on the line:

|---

Best Possible Sleep **Worst Possible Sleep**

How many times do you think you woke up last night: _____ times.

How did last night differ from your usual night's sleep, taking into account that you slept in a different bed, with electrodes, etc.

Any comments or suggestions:

Please mark each line with an 'X'

Going to bed

Asleep quickly — Long time awake

Felt very physically tense — Felt very physically relaxed

No worries on my mind — Many worries on my mind

Many thoughts — No thoughts

Felt very sleepy — Not exhausted at all

Had many physical ailments — Had no physical ailments

Went to bed in a very bad mood — Went to bed in a very good mood

During the night

Frequently awakened — Uninterrupted sleep

No noises — Very noisy

Very comfortable room temp. — Extremely hot or cold

Very comfortable bed — Very uncomfortable bed

Little or no body movement — Tossed and turned all night

Awakened and took and extremely long time to go back to sleep — Awakened but immediately went back to sleep

Lightest sleep possible — Deepest sleep possible

During the night (Continued)

Many thoughts — No thoughts

Felt very physically relaxed — Felt very physically tense

Had many physical ailments — Had no physical ailments

Extremely pleasant dreams — Extremely unpleasant dreams

Many dreams — No dreams

Upon awakening

Woke up long before or after I expected — Woke up exactly when I expected

Woke up extremely tired — Woke up as rested as possible

Had a very hard time awakening — Woke up as easily as possible

Woke up in a very good mood — Woke up in a very bad mood

Remembered extremely unpleasant dreams — Remembered very pleasant dreams

Woke up feeling as physically poor as possible — Woke up feeling as physically good as possible

Woke up with no worries on my mind — Woke up with many worries

Woke up with no thoughts — Woke up with many thoughts on my mind

Put a check mark (✓) in the appropriate column to indicate if you are experiencing any of the following, **right now**.

	Not al All	Slightly	Moderately	Intensely
Headache				
Unsteadiness				
Faintness				
Breathing difficulties				
Chest pain				
Sweating				
Numbness [specify:]				
Flushing				
Chills				
Heart Palpitations				
Sexual feelings				
Hunger				
Bloating				
Nausea				
Gastric fullness				
Abdominal pain				
Feverishness				
Constipation				
Diarrhea				
Urinary problems				
Blurred vision				
Irritated eyes				
Puffy eyes				
Blacking out of sight				
Noise in ears				
Reduced hearing				
Increased taste sensitivity				
Increased smell sensitivity				
Dry mouth				
Thirst				

Use the chart below to indicate the severity of any pains, aches, or stiffness that you may be experiencing right now.

On the chart a check (✓) in the row labelled '0' indicates no discomfort.
a check (✓)in the row labelled '6' indicates the worst possible discomfort.

	Head	Neck	Shoulders	Upper Limbs	Chest	Upper back	Lower Back	Abdomen	Hips	Lower Limbs
0										
1										
2										
3										
4										
5										
6										

Fatigue Scale

Please check (✓) the statement which best describes your present state of physical energy or fatigue.

	1	Full of energy: enough to tackle my usual physical activities.
	2	Energy level is quite high but not at its peak: most physical activities would pose no problem.
	3	Energy level is such that one would prefer to be doing very light or sedentary tasks at this point.
	4	Energy level is adequate for only routine activities at a leisurely pace.
	5	Energy level is such that it would be preferable to rest before doing any routine activity.
	6	Energy level is quite low: would strongly prefer to rest rather than do anything else
	7	Totally physically exhausted: unable to undertake the least activity.

HOW DO YOU FEEL?

CALM ——— IRRITABLE

HAPPY ——— SAD

ENERGETIC ——— SLUGGISH

RELAXED ——— TENSE

STANFORD SLEEPINESS SCALE

Please check (✓) the statement which best describes your state of sleepiness. (Choose only **ONE** statement)

	1	Feeling active, vital, alert, or wide awake
	2	Functioning at high levels, but not at peak; able to concentrate
	3	Awake, but relaxed; responsive but not fully alert
	4	Somewhat foggy, let down
	5	Foggy; losing interesting in remaining awake; slowed down.
	6	Sleepy, woozy, fighting sleep; prefer to lie down
	7	No longer fighting sleep, sleep onset soon; having dream-like thoughts

Appendix E
SLEEP - WAKE QUESTIONNAIRE

Scale: 0 = Never; 1 = Rarely; 2 = Sometimes; 3 = Often; 4 = Always; 5 = N/A

ID	DATE (dd/mm/yy)	HEIGHT	WEIGHT	SEX

INSTRUCTIONS

Read each of the questions carefully and select a scale number that best describes HOW OFTEN YOU HAVE HAD THESE DURING THE PAST **2 MONTHS**. Place the number in the block immediately to the right of the item.
Do not skip any item and print your numbers clearly. Make sure that you have answered all the questions.

SCALE

0 Never
1 Rarely
2 Sometimes
3 Often
4 Always
5 N/A (not applicable)

EXAMPLE

How often do you awaken more than 5 times at night?

Answer:..... 3

(This means that you **often** awaken more than 5 times at night)

1 **Before going to sleep how often do you engage in the following activities:**
a) Read .....
b) Smoke .....
c) Eat a snack.....
d) Watch TV.....
e) Drink tea, coffee, cola .....
f) Drink water, soft drinks .....
g) Listen to music or radio .....
h) Take sleeping pills or tranquillizers .....
i) Shower or bath.....
j) Exercise or take short walks.....
k) Relaxation exercises (Meditation, Prayer) .....
l) Engage in other activities.....

2 **How often does it take you more than 30 minutes to fall asleep?**

3 **How often are you unable to sleep at all?**.....

4 **Before falling asleep, how often do you experience any of the following:**
a) Coughing, breathing difficulties, suffocation.....
b) Feeling hot and sweaty.....
c) Headaches .....

d) Confusion/disorientation
(do not know where you are) .....
e) Tension and worry .....
f) Unpleasant thoughts .....
g) Aches or pains in: .....
limbs .....
neck .....
back .....
chest .....
abdomen .....
h) A need to move your legs because of
unpleasant sensations in them .....
i) Sudden jerking movements of your arms
and legs .....
j) Unable to move arms or legs .....
k) Unable to stop thinking about recent or
past events .....
l) Frightening or strange hallucinations .....
m) Other (specify) .....

5 **How often do you awaken more than 3 times at night?** .....

6 **If you awaken at night, how often do you stay awake more than 30 minutes before you go to sleep again?**.

7 **If you awaken at night, how often do these awakenings happen during:**
a) the first third of the night .....
b) the second third of the night .....
c) the last third of the night .....

8 **How often are your awakenings during the night due to:**
a) External noises (telephone, baby crying
noisy traffic) .....
b) Nightmares or unpleasant dreams .....
c) Aches and pains in different parts of the body
(specify) .....
d) Coughing, choking, breathing difficulties .....
e) Sudden jerking movements of arms and legs .....
f) Need to urinate .....
g) Heartburn .....
h) Headache .....
i) Other (specify) .....

9 **During your sleep at night, how often have you noticed or have you been told that you do any of the following?**
a) Snore .....
b) Turn your head from side to side .....
c) Move your arms or legs or kick .....

d) Talk during your sleep .....
e) Walk during your sleep .....
f) Scream or shout .....
g) Grind your teeth .....
h) Cough.....
i) Suffer from interrupted breathing .....
j) Bed wetting.....

10 **How often is your sleep:**
a) Light .....
b) Deep .....

11 **How often do you have disturbing (bad) dreams during sleep?** .....

12 **When you wake up from these bad dreams, how often do you experience the following?**
a) Feel relieved .....
b) Feel frightened .....
c) Have no emotion at all .....
d) Feel heart pounding.....
e) Breathe heavily.....
f) Choke.....
g) Feel pressure on your chest.....
h) Not able to move .....
i) Feel restless.....
j) Perspire .....
k) Scream .....
l) Wet your bed.....
m) Immediately fall asleep .....

13 **How often do you awaken in the morning feeling:**
a) Good mood.....
b) Bad mood.....
c) Refreshed.....
d) Physically tired.....
e) Headache .....
f) Muscle stiffness or pain in:
limbs.....
neck .....
back .....
chest.....
abdomen .....

14 **How often do you nap during the day:**
a) On work/school days? .....
b) On weekends and holidays? .....

15 **How often do you feel refreshed after a daytime nap?** .....

16 **How often do you fall asleep during the following situations?**
a) While travelling (car, train, etc.) .....
b) In the movies or theatre.....
c) During talks or lectures .....
d) While watching TV.....
e) During social situations.....
f) While reading .....
g) During work.....
h) While driving a car.....
i) While eating.....
j) During other activities (specify) .....

17 **How often do you stop an activity because of an irresistible need to sleep?**

18 **During the day, how often do you:**
a) Feel refreshed and energetic.....
b) Feel physically exhausted and listless.....
c) Yawn.....
d) Have problems at work/school due to sleepiness or naps
e) Have attacks of sudden muscle weakness
or falling.....
f) Have automatic activity (i.e., driving or
walking without recalling where you are) .....
g) Feel faint or lose consciousness .....
h) Feel dizzy or unsteady.....
i) Have unusual sensation (numbness, tingling)
in arms and legs.....
j) Have headaches.....
k) Have pain or discomfort in: limbs.....
neck.....
back.....
chest.....
abdomen .....

19 **How often do you have to work on shifts?**.....

20 **How often do you work on the:**
a) Day shift? .....
b) Evening shift? .....
c) Night shift?.....

21 **How often does your work require you:**
a) to stay awake most of the night? .....
b) to travel from one time zone to another?.....

22 **How often during your work are you exposed to:**
a) Continuous noises?.....
b) Monotonous activity?.....
c) Social isolation? .....

d) Pressures to increase your work output? .....

23 **How often have you used medications for the following purposes? Specify type:**

a) to relieve pain (e.g. aspirin). Specify type... .....
b) to relieve heartburn or indigestion
(e.g. Antacids). Specify .....
c) to diminish nervousness (e.g. Tranquillizer)
Specify.....
d) to relieve depression (e.g. antidepressant)
Specify.....
e) to help you sleep better. Specify .....
f) to keep you awake during the day. Specify .....
g) caffeine tablets .....
h) against allergy (e.g. antihistamines). Specify.....
i) against asthma (e.g. aminophylline). Specify .....
j) to prevent convulsions (e.g. dilantin). Specify.....
k) to treat heart problems. Specify .....
l) to treat respiratory problems. Specify .....
m) to treat high blood pressure. Specify.....
n) hormones. Specify.....
o) to treat Parkinsonism. Specify.....
p) to reduce weight. Specify.....
q) other types of medicines. Specify .....
(1)
(2)
(3)

24 **How often have you used:**

a) Marijuana/hash? .....
b) Cocaine/crack?.....
c) L.S.D., mescaline, ecstasy? .....
d) Stimulants (speed drugs, uppers,
mood elevators, ephedrine)?.....
e) Narcotics (morphine, heroin, opium)?.....
f) Other. Specify? .....

SLEEP - WAKE QUESTIONNAIRE – Part II

INSTRUCTIONS

The following are statements that describe some measurable aspects of your experience. Read each statement carefully and put in the appropriate box the nearest number that describes your experience.

If the statement does not apply to you, put "N/A" on the appropriate line.

1. During work/school days, I usually sleep _____ hours.

2. During weekends and holidays, I usually sleep _____ hours.

3. If I nap, they usually last _____ minutes each .

4. During the past 6 months, I have had _____ nightmares each week.

5. During the past 3 years because of sleepiness : (a) I had _____ work accidents during day time.
   (b) I had _____ work accidents during night time.
   (c) I had _____ car accidents during day time.
   (d) I had _____ car accidents during night time.

6. During the past month, I had to change: (a) from morning shift to night shift _____ times.
(b) from night shift to morning shift _____ times.
(c) from evening shift to night shift _____ times.
(d) from night shift to evening shift _____ times.
(e) from morning shift to evening shift _____ times.
(f) from evening shift to morning shift _____ times.

7. Each day I usually drink: _____ (a) cups of caffeinated coffee.
_____ (b) cups of regular tea
_____ (c) cups of herbal tea, Specify types: _____

8. Each day I usually take: _____ (a) vitamins; Specify _____
_____ (b) herbal remedies; Specify _____

9. Each day I usually smoke: _____ (a) cigarettes.
_____ (b) other; Specify _____

10. Each week I usually drink: _____ (a) glasses of cola.
_____ (b) glasses of wine.
_____ (c) bottles of beer.
_____ (d) ounces of liquor; Specify _____
_____ (e) ounces of other liquor; Specify _____

FAMILY MEDICAL HISTORY

Please check (✓) in the proper space if any of the following items apply to a member of your family

	Son	Daughter	Brother	Sister	Father	Mother	Other (specify)
1 Sleep walking							
2 Screaming during sleep							
3 Very loud snoring in sleep							
4 Daytime sleepiness							
5 Other sleep problems (specify) a)							
b)							
6 Chronic Fatigue							
7 Death during sleep							
8 Mental illness							
9 Psychiatric treatment							
10 Chronic diseases:							
a Cancer							
b Heart diseases							
c Rheumatoid arthritis							
d Diabetes mellitus							
e Other chronic disease							
11 Neurological Diseases:							
a Epilepsy							
b Other							

HEALTH QUESTIONNAIRE

Please check (✓) in the proper space only the items in the following list that apply to you.

		During the Past Year	More Than A Year Ago
		A	B
1	Diabetes		
2	Thyroid disorders		
3	Epilepsy		
4	Psychiatric illness		
5	Psychiatric treatment		
6	Neurologic disease		
7	Kidney disease		
8	Peptic ulcer, gastritis		
9	Intestinal disease (colitis)		
10	Liver disease		
11	High blood pressure		
12	Heart disease		
13	Headache		
14	Arthritis		
15	Back pain		
16	Obesity		
17	Asthma		
18	Pneumonia		
19	Enlarged tonsils, adenoids		
20	Repeated throat infections		
21	Chronic sinusitis		
22	Deviated Nasal Septum		
23	Other health problems (Specify)		
	Hospitalization:		
24	1 or 2 times		
25	3 or 4 times		
26	More than 4 times		
27	Surgery on mouth and/or nose (Specify)		
	For Women Only:		
28	Irregular menstrual periods		
29	Use of birth control pills		
30	Problems associated with menopause		

Appendix F

Letter of Appreciation / Feedback
Brock University Sleep Research Laboratory
Psychology Department

Title of Study: Effects of sleep loss on human performance

Principal Investigator: Kimberly A. Cote, Ph.D.
Co-Investigator: Ryan Renn, B.Sc., Cathy Mondloch, Ph.D., Cheryl McCormick, Ph.D.

Thank you participating in this research program.

If you were in the Sleep Deprivation group, it is pertinent that you go home immediately after the study ends to sleep. You should not drive home yourself as sleep loss impairs driving performance. You should not attempt to work or drive before obtaining enough sleep to feel rested and alert. Specifically, you should go to sleep immediately upon getting home this evening, by 7 or 8 pm at the latest, and sleep through until morning to achieve a sufficient amount of sleep (i.e., get 10-12 hours of recovery sleep). Following the recovery sleep period, you should still exercise the usual caution one would if experiencing sleepiness, e.g., if you feel any residual effects or you were not able to sleep well during the recovery sleep.

The competition game you just played was in fact played with the computer and not another person. The maximum number of points anyone could achieve would result in an award of $10. You will be paid the $10 regardless of your performance. The total honorarium for the study will therefore be $110.

If you need to contact the Sleep Laboratory regarding your participation, or if you have questions in the future regarding any of the study procedures, please call us at **905-688-5550, ext. 3795.** Alternatively, you may contact the Principle Investigator, Dr. Kimberly Cote in the Psychology Department at (905) 6885550, extension 4806.

No individual feedback from the sleep study or performance data may be provided at any time. Feedback about the outcome of the study will be available by request after final publication of the data (email: kcote@brocku.ca).

This research is funded by the Natural Science and Engineering Research Council (NSERC) of Canada. This study has been cleared by the Brock Research Ethics Board (File # 09-033). For answers to questions about your rights as a research participant, contact the Research Ethics Office at (905) 688-5550 ext 3035, or reb@brocku.ca

Below is a more detailed description of the background and rationale for the research study, as well as some of our expected findings.

Scientists, philosophers and poets have long explored the mysteries of sleep and the question of

sleep need. The first laboratory study of human sleep deprivation was carried out by Psychologists over 100 years ago (by Patrick and Gilbert, published in Psychological Reviews in 1896). In that study, researchers monitored three men during 90 hours of wakefulness and observed five important phenomena that are still known to be true today after a century of further research. These observations were: 1. uncontrollable micro-sleeps or naps; 2. dream reports during these sleep periods; 3. mental lapses during performance tasks; 4. fluctuations in performance across the day; and, 5. recovery sleep characterized by deeper sleep.The research that followed over the next 100+ years supported these early findings, and in general, showed that sleep loss reliably leads to deficits in mood, reaction time, attention, and memory. It has been clear for some time that sleep deprivation affects human performance; however, the underlying brain function leading to these deficits remains poorly understood.

Your participation in this study will help us to understand the nature of the effects of sleep loss on mood, alertness, and performance. The Sleep Research Laboratory at Brock University is one of the few centres in the world equipped to investigate brain wave activity during sleep and wakefulness from multiple electrode sites across the scalp (e.g., using the electrode cap that you wore during performance testing). By recording from the multiple sites across the scalp, we can obtain an image or map of your on-going brain activity as it relates to rapid changes in arousal, attention and performance. The performance tasks used in this study were designed to measure cognitive processes that involve activity of the frontal lobe area of the brain and areas that are involved in emotion.

Recent studies using fMRI imaging techniques have shown that the frontal lobe regions of the brain are compromised during sleep loss. In addition, these imaging techniques have shown that our brains are capable of compensation when faced with a sleep loss challenge, that is, the brain adapts by allowing certain brain regions to become active and take-over when the frontal regions fail. By using the unique technology available in the Sleep Research Lab here (e.g., the EEG from multiple sites), we will be able to explore this age-old question of why performance fails when we are sleepy. More specifically, the EEG techniques allow us to monitor real-time activity of the brain (whereas fMRI imaging techniques do not have this sensitivity). In doing so, we aim to be able to understand what brain regions are involved in performance instability during sleep loss, and how the brain compensates for this impairment. In the long-term, this research may allow prediction of human performance failure during sleep loss. This work has widespread implications for improving work and scholastic performance, driving safety, quality of life, and health.

Answers to some frequently asked questions?

Why do you collect so much information about sleep habits, health, and drug-use?
This information is mainly used for screening purposes, to make sure that all participants are healthy, good sleepers. However, some information (e.g., personality) is used to investigate the role of individual differences in response to a sleep loss challenge. For example, some people will be vulnerable to this amount of sleep loss, while others will appear to be relatively resilient. A major area of study in the field of sleep research is to understand the nature of these individual differences.

Why is information collected about phase of the menstrual cycle for women?
While we are not investigating any specific research questions related to phases of the menstrual cycle, some previous research has found that women's sleep varies across the different phases of the menstrual cycle. For this reason, we collect this information to ensure that female participants are run through our study at all phases of the menstrual cycle.

Appendix E

Correct artifact free trials used to generate individual averages for Flanker ERPs

ID	Group	CG	# CG trials	%	IG	# IG trials	%	Error	# Error trials	%
1	C	146	97	66	247	161	65	53	39	74
2	C	142	98	69	249	167	67	117	55	47
3	C	147	112	76	246	195	79	97	84	87
4	C	172	124	72	298	224	75	55	34	62
5	C	124	77	62	210	132	63	100	54	54
6	C	173	99	57	315	204	65	38	24	63
7	C	156	104	67	268	165	62	48	30	63
8	C	156	89	57	276	155	56	40	23	58
9	C	160	108	68	313	208	66	18	7	39
10	C	164	151	92	277	252	91	42	42	100
11	C	160	119	74	304	231	76	39	25	64
12	C	175	100	57	337	220	65	15	10	67
13	C	183	113	62	338	236	70	40	24	60
14	C	159	102	64	283	203	72	83	52	63
15	C	167	117	70	305	208	68	34	22	65
16	C	163	108	66	293	196	67	68	35	51
17	C	142	100	70	228	168	74	39	15	38
18	C	144	102	71	262	180	69	119	92	77
19	C	171	153	89	309	276	89	38	32	84
20	C	158	144	91	288	267	93	77	72	94
21	C	169	132	78	315	257	82	42	26	62
22	C	173	145	84	324	284	88	28	27	96
23	C	162	120	74	288	219	76	75	52	69
24	C	148	117	79	284	224	79	12	5	42
25	SD	148	85	57	270	143	53	62	29	47
26	SD	146	89	61	280	169	60	52	33	63
27	SD	147	90	61	271	170	63	15	8	53
28	SD	144	122	85	290	245	84	46	43	93
29	SD	162	110	68	309	230	74	23	16	70
30	SD	158	62	39	286	109	38	78	43	55
31	SD	142	105	74	250	188	75	109	59	54
32	SD	151	105	70	291	216	74	54	46	85
33	SD	169	146	86	298	238	80	48	42	88
34	SD	118	94	80	206	178	86	75	68	91
35	SD	147	65	44	310	116	37	14	10	71
36	SD	173	128	74	318	254	80	21	21	100
37	SD	134	66	49	227	123	54	56	47	84
38	SD	146	89	61	271	175	65	44	23	52
39	SD	115	89	77	216	175	81	113	94	83
40	SD	174	74	43	313	142	45	31	6	19
41	SD	113	101	89	192	179	93	45	19	42
42	SD	130	64	49	217	98	45	135	70	52
43	SD	157	68	43	255	113	44	80	35	44
44	SD	163	86	53	318	164	52	27	16	59
45	SD	150	91	61	258	162	63	78	32	41
46	SD	159	120	75	232	151	65	126	76	60
47	SD	146	118	81	241	188	78	109	78	72
48	SD	158	66	42	248	100	40	92	32	35

Note: CG = correct congruent trials before artifact rejection; # CG trials = number of artifact free correct congruent trials; IG = incongruent trials before artifact rejection; # IG trials = number of artifact free correct incongruent trials; Error = response locked error trials after before artifact rejection, ; # Error trials = number of artifact free error trials; % = Correct artifact free divided by original trial set before artifact rejection.

Appendix F

Correct artifact free trials used to generate individual averages for Go/NoGo ERPs

ID	Group	Go	# Go trials	%	NoGo	# NoGo trials	%	Error	# Error trials	%
1	C	470	243	52	44	22	50	75	43	57
2	C	470	270	57	62	34	54	57	25	44
3	C	475	307	65	88	55	63	32	24	75
4	C	470	269	57	68	43	63	51	28	55
5	C	470	225	48	53	27	51	67	43	64
6	C	480	273	57	95	49	52	25	6	24
7	C	470	269	57	59	30	51	61	39	64
8	C	470	204	43	47	25	53	69	37	54
9	C	470	388	82	88	69	79	32	25	78
10	C	470	444	94	77	67	87	43	41	95
11	C	432	261	60	66	31	47	51	30	59
12	C	475	230	48	100	47	47	20	7	35
13	C	466	279	60	64	38	60	56	26	46
14	C	451	219	49	54	24	44	63	34	54
15	C	470	222	47	95	53	56	25	17	68
16	C	470	249	53	55	24	43	65	38	58
17	C	456	189	41	62	26	42	57	15	26
18	C	466	237	51	43	23	53	76	47	62
19	C	480	394	82	94	81	87	27	25	93
20	C	466	429	92	79	72	91	41	41	100
21	C	475	398	84	90	80	89	30	28	93
22	C	446	259	58	48	23	48	70	42	60
23	C	470	325	69	85	49	58	35	24	69
24	SD	466	208	45	77	34	44	41	19	46
25	SD	446	341	76	74	45	60	38	35	92
26	SD	446	193	43	74	29	39	43	21	49
27	SD	408	345	85	103	67	65	13	13	100
28	SD	480	278	58	50	33	65	70	38	54
29	SD	432	283	66	38	24	63	72	37	51
30	SD	278	101	36	34	15	45	44	28	64
31	SD	408	326	80	89	69	78	26	24	92
32	SD	384	190	49	54	28	52	49	33	67
33	SD	408	123	30	90	16	18	23	8	35
34	SD	442	334	76	84	60	71	32	27	84
35	SD	413	191	46	46	25	55	64	39	61
36	SD	470	133	28	80	28	35	38	18	47
37	SD	398	263	66	47	39	83	62	46	74
38	SD	475	230	48	70	49	70	50	28	56
39	SD	413	137	33	64	31	49	44	21	48
40	SD	374	179	48	28	18	65	72	47	65
41	SD	446	322	72	50	34	67	64	23	36
42	SD	432	205	47	64	36	57	52	36	69
43	SD	418	170	41	65	23	35	47	14	30
44	SD	456	250	55	58	24	42	59	39	66
45	SD	446	357	80	30	21	70	80	64	80

Note: ID = participant ID; Grp = group; Go = correct Go trials before artifact rejection; # Go trials = number of artifact free correct Go trials; NoGo = NoGo trials before artifact rejection; # NoGo trials = number of artifact free correct NoGo trials; Error = response locked error trials before artifact rejection, ; # Error trials = number of artifact free error trials; % = Correct artifact free divided by original trial set before artifact rejection.